

**A CORRELATIVE STUDY OF CLINICAL PRESENTATIONS  
AND ELECTRO ENCEPHALOGRAPHIC ABNORMALITIES  
IN CASES OF PARTIAL EPILEPSIES**

**THESIS FOR  
DOCTOR OF MEDICINE  
(MEDICINE)**



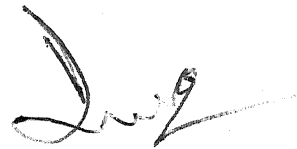
**BUNDELKHAND UNIVERSITY  
JHANSI [U. P.]**



# CERTIFICATE

---

This is to certify that the work entitled "A CORRELATIVE STUDY OF CLINICAL PRESENTATIONS AND ELECTROENCEPHALOGRAPHIC ABNORMALITIES IN CASES OF PARTIAL EPILEPSIES ", which is being submitted as a thesis for M.D. (Medicine) examination, 1992 of Bundel Khand University Jhansi has been carried out by Dr. Bharat bhushan Kathuria under my direct supervision and guidance. The techniques embodied in this work were under taken by the candidate himself. The results and observations recorded were checked and verified by me from time to time.



(D.N.MISHRA)

Dated

M.D., M.N.A.M.S., F.C.C.P.,  
Professor of medicine  
M.L.B. Medical College  
JHANSI

(GUIDE)




## CERTIFICATE

---

This is to certify that the work entitled "A CORRELATIVE STUDY OF CLINICAL PRESENTATIONS AND ELECTROENCEPHALOGRAPHIC ABNORMALITIES IN CASES OF PARTIAL EPILEPSIES ", which is being submitted as a thesis for M.D. (Medicine) has been carried out by Dr. Bharat bhushan Kathuria in the Department of medicine, M.L.B. Medical College, Jhansi. He has put in the necessary stay in the Department of medicine as per University regulations.

Dated



(R.C. ARORA)  
M.D., D.Sc.  
Professor and Head  
Department of medicine  
M.L.B. Medical College  
JHANSI.

## ACKNOWLEDGEMENTS

---

The most pleasant duty in a work like this is to remember and record the contribution of all those without whom the preparation of this would have never been possible.

This has indeed been a rare privilege to have had an opportunity to work under the inspiring guidance of our learned and experienced teacher Dr. D.N. Mishra, MD, FCCP, MNAMS, Professor of Medicine, M.L.B. Medical College, Jhansi. It is to him, I owe a lot in terms of knowledge and experience and the techniques of clinical practice. To him I owe an inexpressible gratitude.

I wish to express my gratitude for the continuous help and inspiration to my teacher Prof. R.C. Arora, M.D., D.Sc., Head Department of Medicine, M.L.B. Medical College, Jhansi. This work could have never been completed, had he not shown the path through the unsurmountable obstacles.

I have no words to express my regards to my esteemed teacher Dr. G.D. Shukla, M.D., Ph.D., MNAMS, Assistant professor (Psychiatry), M.L.B. Medical College, Jhansi, for his keen interest, constant encouragement, timely suggestions, criticism and unlimited help. With my meagre vocabulary, I just can say that it is my profound privilege and pleasure to get the opportunity of expressing my grateful thanks, and almost gratitude to him.

I am equally, grateful to Dr. P.K. Jain, M.D., MNAMS, Dr. Praveen Kumar, M.D., Dip Card., D.M. (Card.), Dr. Navnit Agrawal, M.D., and Dr. T.V.S. Arya, M.D., Assistant Professors, Department of Medicine, M.L.B. Medical College, Jhansi for their constant encouragement to me.

I express my thanks to M/S Universal Computer Systems for their skillful and meticulous computer composing.

Dated ..Oct: 91...

B.B. Kathuria  
(Bharat Bhushan Kathuria)

## CONTENTS

	Page No.
(1) Introduction	1-3
(2) Review of Literature	4-25
(3) Aims and Objectives	26-27
(4) Materials and Methods	28-30
(5) Observations	31-46
(6) Discussion	47-55
(7) Summary and Conclusions	56-59
(8) Bibliography	60-78

oooOooo

# INTRODUCTION

## INTRODUCTION

---

Epilepsies are one of the common neurological disorders which are described in ancient Ayurvedic literature as Apasmara. The word epilepsy is derived from greek words meaning to "seize upon" or " taking hold of".

Epilepsies are an important health problem and second common neurological disorders with a prevalence rate of around 9/1000 in India. So at a given a time, there are about 10 million epileptics in India.

The problem of epilepsy is not simple, because of usually long duration of treatment, attached social stigma and reduction in the working capacity of patient.

By and large the diagnosis of epilepsy is mainly clinical. Epileptology was only at preliminary stage before fifties of this century. But advent of EEG, CT scan and newer antiepileptic drugs has made it compulsory for a Physician to differentiate the idiopathic epilepsies from symptomatic epilepsies and generalised from localization related epilepsies, to ensure precise diagnosis, management and prognosis.

Various attempts have been made by experts to classify seizures and epileptic syndromes. Till date there is no Ideal classification. Most of these classifications are complex, time consuming and require ictal and inter-ictal EEG

recordings. Another difficulty is that the terminology of epilepsies has changed substantially but older terms like grand mal epilepsy and petit mal epilepsy are used by a large section of physicians today.

The various classification given by International league against epilepsy (ILAE) of 1969, 1981, 1985 and proposed revision of 1989 are being accepted all over the world.

Recently newer investigations like position emission tomography (PET), single photon emission computed tomography (SPECT), Magnetic resonance imaging (MRI) and sophisticated EEG related techniques have facilitated the diagnosis and aetiopathogenesis of some epileptic syndromes.

Facilities of newer investigations are not available in most of the hospitals and EEG remains the mainstay for diagnosis of localisation related epilepsies.

In India higher incidence of partial epilepsies is noted as compared to western countries, probably because of greater frequency of CNS infections and birth injuries.

In partial epilepsies specific type and pattern of seizure varies with locale of lesion. Partial Seizures are classified as simple partial seizures, complex partial Seizures and Secondly generalised partial seizures. Epileptiform EEG activity has been reported in 82% cases of partial epilepsies on routine EEG

(Hughes & Greener 1985). EEG May express a partial epileptic seizure by the appearance of repetitive activity from one region which is dissimilar to its background rhythm and which is not due to change in alertness. Attenuation of spontaneous activity has been described as representing seizure onset.



# **REVIEW OF LITERATURE**

## REVIEW OF LITERATURE

### 1) Historical aspects

Epilepsy has the longest medical history (Hock & knight 1947) it was recognised in India before the 10th century b.c. (Joshi, 1971) Epilepsy is defined as "Apasmara" in ancient Indian medical texts. The prefix "Apa" meaning negation or loss & "Smara" meaning recollection of conscious. charak described epilepsy as "paroxysmal loss of consciousness due to disturbance of memory and understanding of mind attended with convulsive seizures.

Epilepsy has been described in greek literature as early as fifth century BC Famous persons in world the Alexander the great, Julius caesar, st paul napolean, lord byron & Maupassant had epilepsy.

By careful pathological & clinical correlative studies Hughlings jakson was first to interpret various manifestations of psycho motor seizures on a sound anatomical basis. He was one of the first to stress that psychic phenomena are as much manifestations of epileptic discharge as the more spectacular tonic & clonic contractions.

### Nomenclature

Various terms has been used, see has falling disease, fits, convulsions, seizure & epilepsy. The term 'Epilepsy' derived

from greek words meaning "to seize upon" or taking hold of. As it is not a single disease, but a group of diseases manifesting in a common way, correct word nowadays in practice is epilepsies or epilepsies and epileptic syndromes'.

To define epilepsies precisely is not easy. Epilepsy are said to be a "tendency to recurring epileptic seizures" (Pefield & Fricksen 1941) it does not seems sufficient. To define epilepsies as paroxysmal cerebral dysarrhythmia is also in sufficient. Hugling Jackson described epilepsy as recurrent seizures due to intermittent derangement of nervous system as a result of sudden excessive disorderly discharge of cerebral neurons. Best possible definition of epilepsies given by dichter (1987) is "Epilepsies are a group of disorders characterised by chronic recurrent paroxysmal alterations in neurological function caused by abnormalities in the electrical activity of brain".

### CLASSIFICATION

As a basis to any discussion on classification of epileptic phenomena we need to be clear about distinctions between classification of epileptic seizures as opposed to those which apply to epilepsies themselves. With seizures we are primarily concerned with clinical manifestations and with their neurophysiological basis, whereas with epilepsies we are more concerned with grouping of seizures or syndromes, their natural histories and their underlying causes. Failure to observe these distinctions has often led to confusion in the past and still seems to be doing so, therefore at present time both of below

mentioned classifications (classification of epileptic seizures 1981 and classification of epilepsies 1989) are relevant.

### CLASSIFICATION OF EPILEPTIC SEIZURES

(Modified from classification of I.L.A.E.1981)

#### PARTIAL SEIZURES (Focal,Local)

##### 1. Simple partial seizures :

- With motor signs
- With somatosensory or special sensory symptoms
- With autonomic symptoms or signs.
- With psychic symptoms.

##### 2. Complex partial seizures (with impairment of consciousness)

- simple partial onset followed by impairment of consciousness
- with impairment of consciousness at onset.

(a) - With impairment of consciousness only.

(b) - With automatism.

##### 3. Partial seizures secondarily generalised.

#### GENERALISED SEIZURES :

##### (1) Non-convulsive seizures :

(a) Absence seizures.

(b) Atypical absence seizures.

(c) Myoclonic seizures.

(d) Atonic seizures.

(2) Convulsive seizures -

(a) Tonic -clonic seizures.

(b) Tonic seizures.

(c) Clonic seizures.

#### UNCLASSIFIED EPILEPTIC SEIZURES.

#### CLASSIFICATION OF EPILEPSIES AND EPILEPTIC SYNDROMES

(Modification of ILAE 1985 classification proposed in 1989 by commission on classification and terminology of ILAE.)

#### A. LOCALISATION RELATED (LOCAL,FOCAL,PARTIAL) EPILEPSIES AND SYNDROMES :

(1) Idiopathic (with age related onset)

- Benign childhood epilepsy with centrotemporal spikes
- Childhood epilepsy with occipital paroxysms
- primary reading epilepsy.

(2) Symptomatic :-

- Chronic progressive epilepsia partialis continua.
- syndromes characterized by specific modes of Preeipitation
- Temporal lobe epilepsies.
- Frontal lobe epilepsies.
- Partietal lobe epilepsies.
- Occipital lobe epilepsies.

(3) Cryptogenic (cause not known)

## B. GENERALISED EPILEPSIES AND SYNDROMES.

(1) Idiopathic :

- Benign neonatal familial convulsions.
- Benign neonatal convulsions.
- Benign epilepsy in infancy.
- Childhood absence epilepsy.
- Juvenile absence epilepsy.
- Juvenile myoclonic epilepsy.
- Epilepsy with generalised tonic clonic seizures (GTCS) on awakening.
- Other generalised idiopathic epilepsies.
- Epilepsies with seizures precipitated by specific modes of activation (Reflex epilepsies).

(2) Cryptogenic

- West syndrome.
- Lennox Gastaut syndrome.
- Epilepsy with myoclonic astatic seizures.

(3) Symptomatic

(1) Non-specific aetiology :

- Early myoclonic encephalopathy.
- Early infantile encephalopathy with suppression bursts.
- Other symptomatic generalised epilepsies.

(ii) Specific syndromes

- Epileptic seizures complicating other disease states.

(C) Epilepsies and syndromes undetermined whether focal or generalised.

(1) With both generalised and focal seizures.

- Neonatal seizures.
- Severe myoclonic epilepsy of infancy.
- Epilepsy with continuous spike and waves during slow wave sleep.
- Acquired epileptic aphasia.
- Other undetermined epilepsies.

(2) Without unequivocal generalised or focal features.

(D) Special syndromes

- Situation related seizures
- febrile convulsions.
- Isolated seizures or isolated status epilepticus.
- Seizures occurring only with acute metabolic or toxic events.

The important modifications in 1989 classification of epilepsies and epileptic syndromes proposed by ILAE are

1- Differentiation between the term "idiopathic" and "Cryptogenic" and inclusion of latter subtype. Idiopathic epilepsy has no underlying cause and is defined by age related onset, clinical and electroencephalographic characteristics and a presumed genetic etiology. The Term cryptogenic refers to a disorder whose cause is hidden or occult.

- 2- Inclusion of primary reading epilepsy as an additional entity in Localisation related Idiopathic epilepsies and syndromes.
3. inclusion of chronic progressive epilepsia partialis continua in the Localisation related symptomatic epilepsies and syndromes.
4. Specific mention of anatomical Cortical seizure Localisation in subgroup Localisation related symptomatic epilepsies and syndromes.
5. Another important inclusion is the category of epilepsies with seizures precipitated by specific mode of activation. The classic example of this group is reflex epilepsies for example-Hotwater epilepsy,eating epilepsy,Startle epilepsy.

### EPIDEMIOLOGY

The over all prevalence rate of epilepsy in general population varies from as low 3-4 per 1000 to as high as 10.5 per thousand.Large variations in prevalence rates are attributed mainly to different criteria for selection of cases.A higher prevalence rate in developing countries is expected due to increase in birth trauma and infections.

In vast majority of studies males tend to predominate presumably due to more frequent head injuries.The commonest age of onset of epilepsies is 0-4 years.The incidence rates are higher within first decade.,somewhat lower in second decade and



then become low. The lowest prevalence rates which usually occur in first decade, increase in second decade and show a decline after 50 years.

### AETIOPATHOGENESIS

Li and Jasper (1961) have clearly demonstrated that the epileptic process consists fundamentally of hyperactive and hypersynchronous neuronal discharges. In chronic epilepsies the recurrent neuronal paroxysms that underlie ictal events are transient expression of a more permanently physiologically disordered cortex.

The typical interictal EEG spike and wave complex reflects the summation of synchronised abnormal neuronal membrane potentials consisting of large paroxysmal depolarization shifts followed by prolonged after hyperpolarization. The depolarization shift results in enhanced neuronal excitation, while after hyperpolarization represents inhibition that may prevent ictal episode. Neurones in the cortical areas surrounding an epileptic focus may demonstrate paroxysmal hyperpolarization only forming an inhibitory zone, which appears to prevent epileptic spread during interictal period.

Despite knowledge of neuronal defects that can destabilize the membrane or interfere with balance of excitatory and inhibitory synaptic activity, fundamental mechanisms underlying spontaneous recurrent seizures in epilepsies remain to be unknown. The neuronal basis of seizure termination is mainly due

to self activating inhibitory machanisms rather than neuronal exhaustion.

Site of origin of generalised tonic-clonic seizures is accepted as thalamic intralaminar system and mid brain spreading to cerebral cortex in bilaterally symmetrical and synchronous manner manifested in EEG as generalised spike and wave pattern. This condition has been termed " Corticoreticular epilepsy ".

In partial epilepsies an epileptic discharge can be assumed to be originating from a particular part of cerebral cortex. In some of the partial epilepsies, epileptic discharges though originating in one hemisphere is reflected as mirror image focus in the contralateral homologous area due to spread through commissural connections. The amplitude of the mirror image focus is usually smaller. So in cases of partial epilepsies, EEG findings are confirmatory in localisation of epileptic foci when findings are limited to specific lobe which is unusual.

Secondary generalization of partial epilepsies occurs through reticulothalamic cortical pathway of spread or through wide spread of discharges to same hemisphere from epileptic foci and spread via commissural connections to contralateral hemisphere.

In cases of complex partial seizures 90% of seizures originate from temporal lobe, usually medial aspect of temporal lobe. In remaining cases frontal lobe is commonly involved. Disturbance in consciousness favours involvement of

centrocephalic region.

Inhibitory neurotransmitter gamma amino butyric acid (GABA) may be a natural anticonvulsant found in the brain. GABA and acetyl choline have opposite effects upon neuronal excitability and an imbalance between these two substances could be predisposing factor for seizure activity (Jurgelsky and Thomas, 1966).

#### AETIOLOGY

Commonly combination of cerebral insult and a genetic predisposition determines the appearance of epileptic seizures.

Genetic factors may contribute in three ways:

- (i) An individual may inherit a low threshold for seizures as in reactive seizures i.e. benign febrile convulsions of infancy and childhood.
- (ii) Specific primary epileptic conditions (Primary epilepsies) autosomal dominant genetic traits has been identified i.e. childhood and juvenile absence epilepsies, partial sylvian epilepsy.
- (iii) Secondary epilepsies i.e Myoclonic epilepsies, phenyl ketonuria.

## ACQUIRED FACTORS

These include congenital lesion, head trauma, infection of brain and its coverings, brain tumours, systemic toxic and metabolic disorders, mesial temporal sclerosis, birth trauma.

The diagnostic role of inter-ictal EEG in epilepsies has been questioned by many authors (Goodin, 1984). Diagnosis of epilepsy is mainly clinical and role of EEG is restricted to confirmation of provisional diagnosis, to classify the seizure disorder, localisation of epileptic foci and guiding prognosis (insome cases) (Githhchley, 1978). In a study by Goodin (1964) 60% of epileptic patients in general population had positive epileptiform activity on EEG. While only 4% of non epileptic has positive EEG.

## LOCALISATION RELATED EPILEPSIES AND SYNDROMES

### 1. Benign childhood epilepsy with centro-temporal spikes.

This type occurs in 15-20% of epileptics in the age group of 3-13 year, more commonly in males (60%). Seizure pattern consists of brief spells of simple partial hemifacial motor seizures, frequently during sleep (70%), with somatosensory onset and tendency to tonic, tonic-clonic seizures of facial and neck muscles, manifesting as speech arrest and drooling of saliva, seizure, occurs usually once in 2-12 months. EEG changes consists of blunt high voltage centrottemporal spikes often followed by slow waves usually precipitated by sleep.

It is an autosomal dominant gene with age dependent penetrance . Monotherapy with phenytoin or carbamazepine is successful. Usually good recovery occurs before 15-16 years of age.

## 2. Childhood epilepsy with occipital paroxysms.

Onset is usually between 1½ years to 8 years, usual symptoms (amaurosis in 65%, Scotoma in 55% , hallucinations in 25%, illusions in 10% cases) predominate, nocturnal partial seizures (usually hemiclonic) and post ictal headache (30%) are fairly common.

Paroxysmal high voltage spike wave and/or sharp waves localised in the occipital or posterior temporal region over one or both hemispheres occur rhythmically on EEG.

These finding tend to disappear after 9 years of age. Prognosis is favourable regardless of age, seizures are well controlled in 60% cases. Drugs useful are phenytoin, carbamazepine and primidone.

## 3. Benign partial epilepsy with frontal foci:

Partial seizures usually begin between 4-8 years of age. The seizure pattern is of adersive type with head deviation, simple absence seizures may precede or coexist. EEG findings consists of unilateral or bilateral frontal foci. In about 50% cases 3 Hz spike and wave complexes are observed, EEG abnormalities normalize before the age of 13 years.

#### 4. Primary Reading Epilepsy

##### Symptomatic:

Chronic progressive epilepsia partialis continua :

It is seen in young children with unilateral chronic cerebral inflammatory disorders or in adults after severe anoxia and major cerebrovascular accident. The continuous partial motor seizures usually involving face reflect multiple rather than single lesion. This form is often unresponsive to medications. Seizures may disappear after about 1-2 years in about one third of cases.

##### Temporal lobe epilepsies :

This term is not synonymous with complex partial seizures, psychomotor and limbic seizure. The term temporal lobe epilepsies means that a temporal lobe focus in EEG is demonstrable. Symptomatology usually consists of an aura, psychic symptoms, alteration of consciousness automatisms and commonly secondary spread to manifest as tonic-clonic seizures.

Auras consists of experiential phenomena, which patient perceives as warning of impending seizure episode. Psychic symptoms consist of :

- a. dysmnestic symptoms i.e. feeling of familiarity (de ja vu) and unfamiliarity (Jamais vu)
- b. cognitive disturbances such as dreamy state, depersonalization and time distortion.

c. Affective symptoms, fear and rage, depression and elation and sometimes sexual excitation (orgasmic epilepsy).

d. Delusions and hallucinations.

Alteration of consciousness unusually reflects involvement of limbic structures and their connections. Autonomic auras such as epigastric distress or vomiting most commonly precede temporal lobe epilepsies which are associated with altered consciousness. Those seizures which are preceded by olfactory aura's are called uncinate seizures. EEG findings consist of unilateral or bilateral spikes.

#### Frontal lobe epilepsies.

Seizures pattern may consist of :

a. Tonic, clonic movements starting from discrete parts such as feet, hands and facial muscles and progressing leading to unilateral or bilateral involvement. When spread occurs in an orderly fashion along the precentral gyrus jacksonian march occurs. These seizures may be followed by post ictal weakness (Todd's paresis).

b. Complex versive movements in form of, turning of head, eyes and body towards one side and posturing of one or more extremities.

c. Adversive seizures : Turning of head and eyes away from epileptic focus and elevation of contralateral arm. It occurs with involvement of supplementary motor cortex.

d. Inappropriate laughter associated with humour (Gelastictic epilepsy).

e. Some times they may manifest as psychomotor seizures and atypical absence seizures.

EEG usually shows unilateral or bilateral spike wave complexes and sharp waves.

#### Parietal lobe epilepsies :

They usually manifest as localised paresthesia or numbness from onset of a seizure. Spread occurs readily to frontal cortex leading to sensory motor seizures. Post ictal phenomena such as anaesthesia may follow after simple partial seizures involving parietal lobe.

#### Occipital lobe epilepsies :

The seizures pattern usually manifest as unformed luminous vision and amaurosis.

### GENERALISED EPILEPSIES AND SYNDROMES

#### 1. Benign Neonatal familial convulsions :

They usually occur on 2nd or 3rd day. Seizures pattern consists of clonic or apneic seizures. No specific EEG changes are observed. About 14% of these develop some form of epilepsy.

#### 2. Neonatal convulsions.

They occur on 5th day of birth. Seizure pattern is of clonic or apneic type. Alternate sharp theta waves are observed on EEG.



### 3. Benign myoclonic epilepsy in infancy :

Age group involved is 1-2 years. Seizures pattern consists of brief spells of generalised myoclonus and brief bursts of spike wave complexes are observed on EEG.

### 4. Childhood absence epilepsy (Pyknolepsy)

It affects children 6-7 years of age, manifests as many absence seizures during a day. EEG findings consist of bilateral synchronous spikes and wave. Some of these children may develop Generalised tonic clonic seizures (GTCS) later on.

### 5. Juvenile Absence :(Petit mal).

It manifests as absence with retropulsive movements, seizure frequency is less than childhood absence. EEG findings show 3 Hz spike-wave bursts. This type of epilepsy has excellent response to therapy.

### 6. Juvenile myoclonic epilepsy :(Impulsive petit mal)

It manifests as bilateral single or repetitive arrhythmic irregular myoclonic jerks in arms and shoulders. It may progress to generalised Tonic clonic Seizures associated with infrequent absences. EEG findings consist of precipitation of episode by photic stimulation, rapid generalised 4 to 6 Hz spike waves/polyspike -wave. It affects about 7% of adolescent epileptic patients. Sodium valproate is the drug of choice.

### 7. Epilepsy with Generalised Tonic-Clonic Seizures on awakening :

It usually affects young adults. Familial occurrence is

frequent. Seizure type consist of Generalised Tonic Clonic Seizures shortly after awakening (90%) or in the evening (10%) EEG findings consist generalised high voltage sharp waves following by slow waves.

8. Epilepsies with seizures precipitated by specific modes of activation :

Classic example of this group are reflex epilepsies which are generalised i.e. Hotwater epilepsy, eating epilepsy, tactile epilepsy, paroxysmal dystonic seizures.

### Cryptogenic

#### 1. West syndrome :

It affects infants usually in 4-7 months age group. Seizure type consist of salam attacks. EEG shows burst suppression with distorted background activity. Prognosis is poor, even after treatment with steroids.

#### 2. Lennox Gastaut Syndrome :

Children of 1-8 year of age group show this picture consisting of more than one variety of generalised seizures, presence of mental retardation and generalised sharp and slow wave complexes of less than 3 Hz and some times sleep bursts of fast rhythm.

Refractoriness to common anti-epileptic drugs is the rule.

### 3. Epilepsy with myoclonic astatic seizures :

Age of onset is usually 7-12 years. Frequently male Children are affected. Seizure type consist of myoclonic akinetic absence with clonic and atonic components. Frequently these patients may land into status epilepticus of generalised tonic -clonic type. Regular fast spike wave and polyspike wave abnormalities are noticed on EEG.

### Epilepsies and Syndromes undetermined whether focal or generalised

#### 1. Epilepsy with continuous spike wave activity during sleep.

Seizure type consist of Generalised tonic clonic seizures during sleep and atypical seizures. Observed EEG findings consist of continuous diffuse spike-wave during sleep.

2. Acquired epileptic child hood aphasia (Landau Kleffner syndrome) Seizure type consist of verbal auditory agnosia with secondarily generalised partial motor seizures. EEG findings consist of multi focal spikes and spike wave complexes. Course is benign usually with complete remission before the age of 15 years.

### EEG in the Diagnosis of epilepsy

Definitive diagnosis of epilepsy requires demonstration of ictal EEG changes simultaneously with occurrence of seizure, which is rarely possible except in patients with absence seizures, juvenile myoclonic seizures and partial seizures with minor motor seizure.

Due to paroxysmal nature of the disease a normal EEG does not exclude epilepsy. The probability of positive EEG findings is related to several factors.

- a. Duration of recording.
- b. Wake-sleep study.
- c. Activation procedures used.
- d. Times of EEG recording.

In a single routine EEG, positive findings are usually observed in around 60% cases with sleep deprivation, but if 3 EEG recordings have been taken epileptiform changes may be observed in 90% cases.

- e. Use of additional electrodes.
- f. Anticonvulsant therapy.

#### EEG abnormalities in epilepsies :

They can be grouped in three categories-

- 1. Non-specific abnormalities
  - 2. Specific abnormalities
  - 3. Background abnormalities
1. Non-specific abnormalities

- a) 3 Hz Delta paroxysms
  - i. Frontal intermittent rhythmic Delta activity (FIRDA).
  - ii. Occipital intermittent rhythmic Delta activity (OIRDA)
- b) Runs of bilaterally synchronous delta.
- c) Bursts of bilaterally synchronous theta.

d) 8-12 Hz activity of higher amplitude than the background alpha activity.

## 2. Specific epileptiform abnormalities.

- |                             |                           |
|-----------------------------|---------------------------|
| a. Spikes                   | b. polyspikes             |
| c. High voltage sharp waves | d. spike-wave complexes.  |
| e. polyspike-wave complexes | f. sharp wave discharges. |

## 3. Background abnormalities.

- a. mild degree of diffuse slowing while on anticonvulsant therapy.
- b. Diffuse marked slowing-may occur during post ictal period for few hours to 1-2 days.
- c. background slowing other than above usually correlates with mental subnormalities.

Following newer investigation have facilitated the diagnosis of epilepsies.

## A) NEUROPHYSIOLOGICAL INVESTIGATIONS :

1. Routine EEG
2. Ambulatory cassette recording
3. EEG telemetry
  - a. Cable telemetry.
  - b. Radio telemetry.
4. EEG recording with closed circuit television by split screen technique.
5. Computerised EEG neuromapping.
6. Magnetic encephalography.

## B) NEURORADIOLOGICAL PROCEDURES :

1. Plain radiography.
2. CT scanning.
3. MRI imaging.
4. Isotope encephalography
5. PET scanning.
6. SPECT scanning.

EEG abnormalities reported in partial epilepsies are sequential spikes and sharp waves, Sinusoidal waves Rhythmic waves and attenuation of spontaneous activity. In a study designed to assess EEG morphology of partial epileptic seizures. Warren T Blume et al (1984) studied 66 consecutive patients with electrographic partial seizures. Different EEG phenomenon noted were

1. Attenuation, sinusoidal waves.
2. Spikes, spike-waves, polyspike-waves, sharp waves, saw toothed waves.

The second group was referred as repetitive epileptiform potentials (REPs).

They noted : Total patients = 66

Sinusoidal waves in 20 patients (30%)

REPs in 17 patients (25%)

Both in 29 patients (45%)

ONSET AND MORPHOLOGY OF EEG CHANGES

Onset	Evolution
Sinusoidal (41)- Sinusoidal only 31	Sinusoidal only 20
REPs - REPs only -25	REPs appear (11)
	REPs only 17
Both (sinusoidal & REPs) -10	Sinusoidal appear-8

Spikes and sharp waves were the most common repetitive epileptiform potentials encountered.

Attenuation of spontaneous activity with onset of seizure was noted in 11% of cases. Gibbs (1964) reported 80% incidence of temporal foci in clinical psychomotor epilepsy.

Many workers have tried to develop methods of deriving components of local activity in certain area. Bo Hjorth (1976) made an important contribution by formulating source sensing derivations connected between EEG amplifiers and recording channels, derivations of this type constitute better description of local activity than do the conventional, common reference and bipolar derivations.

The model implied that the observed surface pattern is a secondary effect of primary field components perpendicular to surface.

Smith et al (1985) have demonstrated the reliability of a non invasive method for successfully localising the local components of scalp EEG. Dipole localisation method (DLM) is a computer assisted, mathematical method based on electrical field theory.

## **AIMS AND OBJECTIVES**



## AIMS AND OBJECTIVES

---

Clinical manifestations of epilepsies are varied and diagnosis of specific type of epileptic state or syndrome is based mainly on history, clinical features, and EEG.

Role of surface EEG is thus restricted to differentiate epilepsy from other causes of seizures, to substantiate the clinical diagnosis of epilepsy, to classify seizure disorders, localising the epileptogenic focus and guiding the prognosis.

In a developing country like India, sophisticated facilities helpful in localisation of epileptogenic source (CT Scan, MRI, PET, SPECT) are available in few centres only. Further a large section of population of developing country like India can not bear the expenses for these investigations. In this context EEG is very cheap and available in most of medical college hospitals. Considering this fact present study was conducted with following aims and objectives :

1. To assess the prevalence of partial (localisation related) epilepsies amongst all epilepsies.
2. To Assess the frequency of different types of localisation related epilepsies.

3. Evaluation of EEG abnormalities in localisation related epilepsies.

4. Study of clinical manifestations of different types of localisation related epilepsies.

5. correlation of specific EEG abnormalities with different types of localisation related epilepsies.

6. To assess the role of scalp EEG in localizing the site of epileptogenic focus.

## **MATERIALS AND METHODS**

## MATERIAL AND METHODS

The patients suffering from seizure disorders who attended the out patient departments of Medicine, pediatrics, Neurology and Psychiatry, of Maharani Laxmi Bai Medical College, Hospital Jhansi from Jan. 1990 to Jan., 1991 formed the sample of the study. Patients who required investigation or having uncontrolled seizures were admitted. Patients of both sexes and all the age groups were included in the study.

### METHODS :

All the patients were evaluated on following lines.

#### Clinical History

1. General History with special emphasis on detail of seizure disorder was taken.
2. Clinical examination, specially of nervous system was performed and a record of clinical symptomatology with clinical examination was maintained.

#### Investigations

1. Routine EEG,

EEG, utilizing 10-20 system of electrode placement with both referential and bipolar recordings, after overnight sleep deprivation with at least 3 minutes of hyperventilation and photic stimulation was recorded for at least 30 minutes on 8 channel EEG machine.

## 2. Radiological :

- I. X-ray skull A.P.skull A.P.and lateral view.
- II. X-ray chest PA view(whenever relevent)
- III. Computerised axial tomography of head if possible.

## 3. Fundus examination.

## 4. Cerebrospinal fluid examination

## 5. Biochemical tests

- a. Serum calcium.
- b. Blood urea,blood sugar.

## 6. V.D.R.L. test.

History of seizure disorder was taken from patient and relatives of partient.Special emphasis was given to :

- i. Age of onset of seizures.
- ii. Frequency of seizures.
- iii. Time of last seizure episode.
- iv. Predisposing and aetiological factors.
- v. Family history of seizures.
- vi. Precipitating factors.
- vii. Generalised or partial seizures.
- viii. Premonitory symptoms.
- ix. Pre-ictal, ictal and post ictal events.
- x. Specific abnormalities.

All the patients were specifically evaluated for the above mentioned data.

General examination and examination of nervous system was interpreted for associated disease with seizure disorder, associated neurological deficit and evidence of localising neurological deficit and evidence of localising value of seizure.

EEG was interpreted using EEG diagnostic criteria given by kiloh et al (1982). regarding presence of

1. Background rhythm
2. Clear paroxysmal (epileptiform events on EEG)
3. Slow wave abnormalities.
4. Sequential spikes, spikes and waves originating from a focus.
5. Symmetry.
6. Presence of rhythmic waves.
7. Attenuation of spontaneous activity at onset of seizure.
8. Sinusoidal waves.
9. Specific abnormalities.

Other investigations, which were required were done in each case. CT scan was done whenever possible.

EEG data obtained from above observations was correlated with different types of localisation related (partial) epilepsies and comparison with similar observations were made.

In the end, findings were tabulated and analysed,

# **OBSERVATIONS**

## OBSERVATIONS

---

Present study was carried out on 196 patients presenting with complaint of seizure/seizures who attended the out patient department of medicine, Paediatrics, Neurology and Psychiatry of Maharani Laxmi Bai Medical college Hospital Jhansi from Jan 1990 to Jan 1991.

Amongst 196 epileptics, 9(4.6%) cases were of unclassified epileptic seizures :-

### Distribution of classifiable cases :-

(a) Seizure type :- Primarily generalised seizures were present in 108 cases (55.10%). 79 cases were of partial seizures. Secondly generalised partial seizures were present in 36 cases followed by simple partial seizures (14 cases) (table I)

Table I: Showing Clinical types of Seizures.

Types of Seizures	No. of cases	Percentage
(A) Generalised Seizures.	108	55.10%
Tonic-clonic	95(87.96%)	
Tonic	2(1.85%)	
Atonic	3(2.77%)	
Myoclonic	2(1.85%)	
Absenses	4(3.70%)	
Atypical Absenses	1(0.92%)	



(B) Partial Seizures	79	40.40%
(i) Secondly Generalised	36	18.36
(ii) Simple Partial	29	14.79
(iii) Complex Partial	14	7.14
Unclassified epileptic Seizures	9	4.59

---

Total	196	100.00%
-------	-----	---------

---

(b) Age of onset :-

In this study age of onset of seizures was highest (48%) in the first decade of life. In 92% cases seizures started below the age at 20 years (Table II)

Table No.II:Showing Age of onset of Seizures

Age Group	No. of cases	Percentage
0 - 10	94	47.95%
10 - 20	86	43.87%
20 - 30	8	4.08%
30 - 40	3	1.53%
40 - 50	3	1.53%
50 - 60	2	1.02%
Total	196	100.00%

---

Out of 196 cases 138 were male and 58 female. Thus Male to Female ratio was 2.38/1. Male predominance has been noted in earlier studies also.

### Features of Partial epilepsies

Nearly 40% of patients in this study presented as Partial Seizures. Age of these patients ranged from 6 months to 52 years Male to Female Ratio in these cases was 2.56/1. Out of these cases, 45.5% (36 cases) presented as Secondarily generalised Partial Seizures, 17.72% (14 cases) presented as Complex partial Seizures and 36.71 (29 cases) presented as Simple Partial seizures. (Table No. III)

Table III :- Various types of partial Seizures

Type of Partial Seizures	No. of cases	Percentage
(A) Simple Partial Seizures	-29	36.7%
(B) Complex Partial Seizures	-14	17.7%
(C) Secondarily generalised	-36	45.6%
Partial Seizures		
Total No. of cases	79	100%

Frequency of Seizure episodes varied from single episode in a year to several episodes per day. 13 patients had only one seizure episode at the time of EEG recording. 7 Patients were admitted in status epilepticus. One patient presented as chronic epilepsia partialis continua.

Predisposing factors could be worked out in 19 cases. Head injury was the commonest cause (9 cases), followed by Birth anoxia - 5 cases and intracranial space occupying lesion (3 cases). Inflammatory brain disease was present in 2 cases.

Precipitating factors were present in 12 cases in form of sleep - 5 cases, sleep deprivation 3 cases, fatigue 2 cases and fever in 2 cases.

Out of 79 cases of partial seizures abnormal EEG findings were found in 58 cases. In 4 cases EEG findings were generalised without any focal changes. In the rest 54 cases hemispherical assymetry on EEG was noticed. Abnormal EEG findings were confined to one hemisphere in 16 cases. Commonest EEG findings in Partial Seizures were sharp waves.

#### Features of Simple Partial Seizures

Amongst total 196 cases, 29 cases (14.8%) had simple partial Seizures. Age of patients ranged from 10 Months to 52 years. Male to Female Ratio was 2.2 :1. In more than half of cases seizures started below the age of 10 years. Predisposing factors could be detected in 4 cases two patients had head injuries, one patient had intracranial space occupying lesion and one case had history of prolonged labour with forceps applications & birth anoxia.

One patient had facial palsy. One patient had right sided hemiparesis with difficulty in speech.

Seizures started on right half of body in 12 cases and left half of body in 16 cases. Postictal features were present in only 20 cases. Clinical features are summarised in Table IV.

Table No.IV:

Clinical features of simple Partial Seizures.

Total No. of cases -29

---

Clinical features.

---

(A) ONSET

Right half of body.

-- Motor	---- 7
-- Sensori Motor	---- 2
-- Sensory	---- 3

Left side of body.

-- Motor	---- 9
-- Sensory	---- 5
-- Sensory Motor	---- 2

(B) ICTAL features

Tonic	---- 13
Clonic	---- 9
Tonic-Clonic	---- 5
Autonomic	---- 2

(C) Postical features (20 cases)

(1).Headache	---- 10
(2).Postictal Paresis	--- 4
(3).Drowsiness	---- 7
(4).Sleep	---- 3
(5).Vomiting	---- 2
(6).Generalised weakness	---- 3

---

Abnormal EEG findings were observed in 16 cases out of 29 cases.

Localisation of EEG abnormalities to a single cerebral hemisphere was possible only in 7 cases. Localisation was not possible in 2 cases. (Table V).

Parietal Lobe was the commonest site of epileptic foci (8 cases) (Table no. VII). EEG abnormalities consisted of sharp waves, spike with sharp waves, and high voltage sharp waves.

Commonest EEG abnormalities observed were repetitive epileptiform potentials (REP's) observed in 11 cases out of total 16 cases in which EEG was abnormal (Table no. VI). Sharp waves were most common. High voltage sinusoidal waves were observed in 5 cases. These waves were generalised with high voltage in specific region in 3 cases and localised in 1 case. Focal slowing (in parietal region) was observed in one case. This patient had a seizure episode 12 hours back. Frontal intermittent rhythmic delta activity was observed in one patient aged 1 1/2 years with history of simple partial seizures after Head injury in frontal region one month back. EEG morphological characteristics had little correlation with different types of simple partial seizures but topographical distribution of EEG abnormalities correlated well with site & type of seizure onset.

CT scan was performed in one patient with history of simple partial seizures (motor) 10 months after head injury. CT scan was normal in this case.

Table No. V :  
Showing EEG features of Simple Partial Seizures

EEG Findings	No. of cases	Percentage
(A) Normal	13	44.83%
(B) Abnormal	16	55.17%
1. Localisation possible	7	
Right Hemisphere	3	
Left Hemisphere	4	
2. Localisation with secondary generalization.	7	
Right Hemisphere	2	
Left Hemisphere	3	
Central Hemisphere	1	
Centroparietal	1	
3. Localisation not possible (Generalized)	2	

Table No. VI

Various EEG abnormalities in simple partial seizures

EEG findings	No. of cases
A) Specific epileptiform Abnormalities	14 cases
i) High voltage sinusoidal waves	5 "
ii) Sharp waves	6 "
iii) Localised spikes	1 "
iv) Spike wave	2 "
v) Sharp waves discharges	2 "
B) Nonspecific abnormalities	
Frontal intermittent	
Rhythmic Delta activity (FIRDA)	1 "
C) Background abnormalities	
Focal slowing	1 "

Table No.VII :

## Epileptic foci in case of simple partial seizures

EEG Focus	No. of cases
1).Frontal	1
2).Parietal	3
3).Central	1
4).Centroparietal	1
5).Parieto temporal	2
6).Fronto temporal	2
7).Occipito parieto temporal	2
8).One Cerebral Hemisphere	2

Features of complex Partial Seizures

Out of 196 cases, there were only 14 cases of complex Partial Seizures. Age of Patients ranged from 7 years to 52 years. In 6 cases seizures started below the age of 10 years in the rest 8 cases seizures started after the age of 10 years. Family history was positive in one case. Predisposing factors could be detected in 3 cases only, two with history of birth anoxia and one with history of head injury. One patient was not performing well in his studies after seizures had started.

Out of 14 cases history of aura was present in 9 cases. Automatisms and motor phenenmenon were present in 8 cases. In 3 patients ictal episode progressed to generalised tonic-clonic

seizures while in two patients ictal episode progressed to generalised tonic seizures.

Post convulsive features were present in 10 cases in form of amnesia (6 cases) Headache 3 cases and vomiting in one. Table VIII summarises the clinical features in cases of complex partial seizures.

Table VIII :Showing Clinical features of Complex Partial seizures (Total no of cases -14)

Clinical Features	No.of cases	Percentage
AURA.	9	64.29%
Visual Hallucinations	2	
Semato sensory Hallucination	3	
Auditory Hallucination	2	
Vertigo	1	
De Javu	1	
Automatisms/Motor Phenomena	8	57.15%
Restlessness	3	
Lateral deviation of Head and eyes.	2	
Upward deviation of eyeballs	1	
Smacking of lips	1	
Tearing of clothes	1	
Amnesia	6	42.85%
<u>Other associated features</u>	5	35.71%
Generalised Tonic-Clonic Seizures	3	
Tonic spasms	2	



## EEG Findings.

EEG abnormalities were observed in 8 cases out of total 14 cases of complex partial seizures. Rest 6 cases had Normal EEG. They consisted of temporal spikes, phase reversal and burst of sharp waves. Bitemporal spikes were present in 2 cases.

In three cases epileptic foci was unilateral without any generalized findings. In 3 cases findings were generalised with some focal change in one side EEG findings are summarised in table No. IX & X.

Table IX: EEG findings in complex Partial Seizures.

Normal	---	6	42.85%
Abnormal	---	8	57.15%
Unilateral temporal Spikes -		3	
Bitemporal Spikes	---	2	
Temporoparietal	---	1	
Frontotemporal	---	1	
Centrottemporal	---	1	

Table X :- EEG findings in complex Partial Seizures.

EEG findings.	No. of Cases.	Percentage.
Normal	6	-42.85%
Abnormal	8	-57.15%
Focal (1) Right	----- 2	
(2) Left	----- 1	

(3) Bilateral ----- 2

Focal with secondarily generalised

(1) Right ----- 1

(2) Left ----- 2

---

Total No. of Cases	14	100.00
--------------------	----	--------

---

C T Scan could be performed in 2 cases only. In one case Bilateral temporal lobe atrophy was present. Another C T Scan was normal .Other investigations were normal.

#### Features of secondarily Generalised Partial Seizures.

Out of total 196 cases studied 36(18%) cases were of secondarily generalised Partial seizures.Age of patients ranged from 1 year to 52 years .Male to female ratio was approximately 201. Frequency of seizure episodes varied from one episode in a year to several episodes in a day.Seven patients had only single seizure episode at the time of recording.Three patients were admitted in status epilepticus.

Predisposing factors could be worked out in 9 cases.Head injury was commonest in 4 cases,followed by history of birth anoxia in 3 cases and intracranical space occupying lesion in 2 cases.Precipitating factors were present in 4 cases in form of sleep(2 case),fatigue and fever (1 case).

Premonitory symptoms were present in 16 cases. They include abnormal sensations, ghabrahat, feeling of muscle contractions in parts where seizures started and headache onset of seizures was from right half of body in 13 cases and from left half of body in 16 cases. In 7 cases patient or patient's attendants were unable to tell exactly the side involved first either due to single episode or due to lack of any person watching the patient at the time of seizures. Common parts of body from where seizure started were, great toes, hands, lips and eyelids. In nearly half of cases patient experienced some sort of abnormal feeling in the part of body from where seizures started well before the onset of seizure episode. Nature of seizure episode at onset was motor, sensory motor, sensory or autonomic.

Motor symptoms were in form of twitching, tonic spasms or clonic movements. Sensory symptoms were in the form of numbness and tingling sensations in the extremities. One patient had voiding of micturition at the onset of seizure and one had vomiting with abnormal sensation in epigastric regions. 28 patients had unconsciousness, while 8 remained conscious. Tonic-clonic movements were present in 30 cases while only tonic spasms in 6 cases.

Post convulsive symptoms were present in form of Headache, drowsiness, post ictal paresis, weakness and confusional states.

Clinical features of secondarily generalised partial seizures are summarised in Table No.(XI).

Table No.XI :

Clinical features of secondary generalised epilepsies  
(Total No. of cases 36)

Clinical features		No.of cases	Percentage
ONSET			
Side	Right	13	36.11%
	Left	16	44.44%
	Could not be confirmed	7	19.44%
	Motor	16	44.44%
	Sensori motor	10	27.77%
Nature			
	Sensory	8	22.22%
	Autonomic	2	5.55%
CONVULSIVE PHASE			
	Unconsciousness	28	77.78%
	No alteration of consciousness	8	22.22%
	Tonic-clonic movements	30	83.33%
	Tonic spasms	6	-16.67%
POSTCONVULSIVE PHASE.			
	Headache	10	
	Headache & Drowsiness	22	
	Postical Paresis	8	
	Generalised weakness	2	
	Confusional state	2	

Associated Neurological features were present in 10 cases which are summarised below in table XII.

Table No.XII :

Neurological features of Secondarily Generalised epilepsy.

Clinical	No. of cases
Mental Retardation	3
Hyperactivity	1
Optic Atrophy	1
Hemiparesies	3
Transient Aphasia	2
Total	10

#### EEG findings

Out of 36 cases of secondarily generalised partial seizures abnormal EEG was found in 29 cases (80.5%). In 4 cases EEG findings were generalised without any focal changes. In rest 7 cases EEG findings were bilaterally assymmetrical on two sides. In 18 cases findings were generalised with some focal changes on one side.

EEG findings were in form of high voltage sinusiodal waves, Sharp waves, Burst of sharp waves, phase reversal, spikes, spike and waves.

Table No.XIII :

## EEG findings of secondarily Generalised Partial Seizures

(A) Normal	7	19.44%
(B) Abnormal	29	80.56%
(i) Generalised	4	
(ii) Focal	7	R-3
(iii) Secondarily Generalised with focal	18	L-4
-- Right Hemisphere	6	
-- Left Hemisphere	8	
-- Central	3	
-- Bilateral	1	

As is shown in Table XIII, 29 cases showed abnormal findings. In 4 cases EEG findings were generalised and no focal changes were present. In 7 cases findings were confined to one or another hemisphere.

In 18 cases an epileptic focus was present with generalised epileptiform abnormalities on EEG. Among these findings were confined to right hemisphere in 6 cases, to left hemisphere in 8 cases, to central region in 3 cases and bilateral in one case.

In 25 cases an epileptic focus was present. In nine cases epileptic focus involved one area of scalp. In 16 cases epileptic

focus was not exactly Localised and involved two areas of scalp. In four cases localisation of epileptic foci was not possible because changes were generalised. (Table XIV)

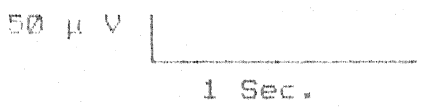
Table No.XIV :

Showing Focus in cases of secondarily generalised seizures

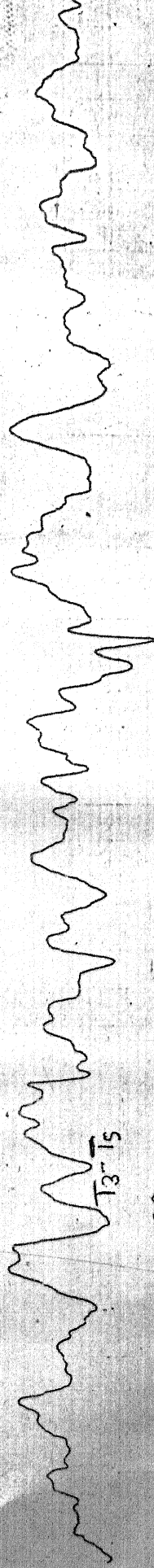
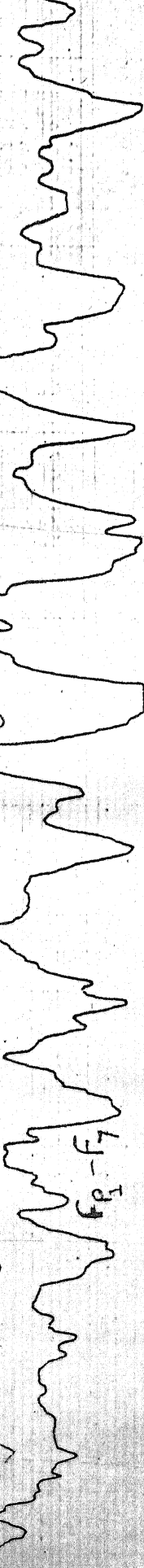
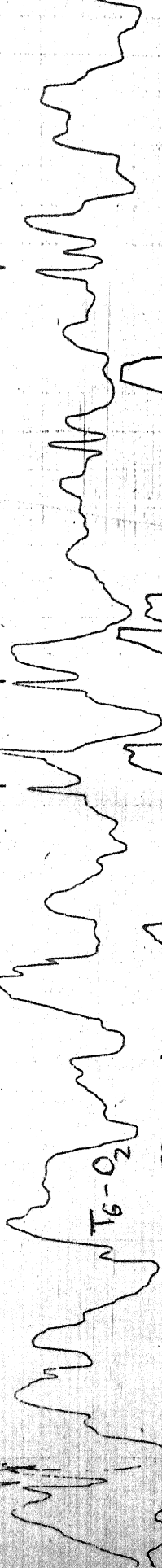
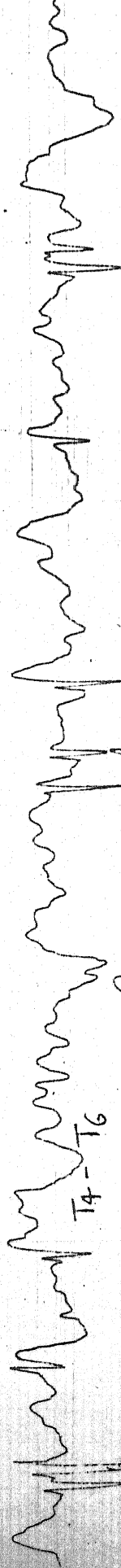
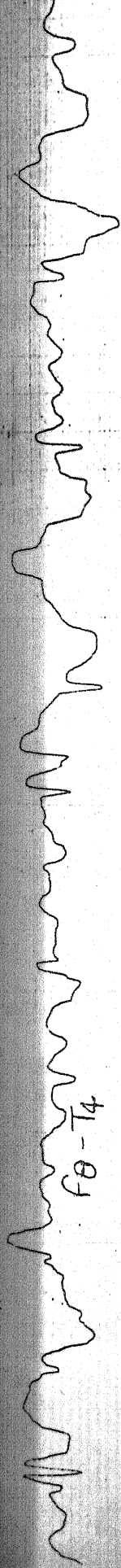
EEG Focus		
(1) Frontal	2	
(2) Parietal	4	
(3) Temporal	2	
(4) Occipital	1	
(5) Fronto parietal	2	
(6) Centro parietal	6	
(7) Occipito parietal	1	
(8) Parieto temporal	2	
(9) Occipito temporal	4	
(10)Bitemporal	1	
<hr/>		
(A) Localisation possible (In above cases)	25	(69.45%)
(B) Localisation not possible	4 cases	
(C) Normal EEG	7 cases	
<hr/>		
Total No. of cases	36	100.00%

CT Scan was performed in 3 cases one patient showed a granuloma in left parietal region. In one case it showed a tuberculoma in central region. In rest of the cases CT Scans were normal. On examination of fundus of one patient optic atrophy was detected. X-Ray chest of one patient showed bilateral tubercular infiltration. X-Ray skull were normal. Other investigations were normal.

EEG No. 532 : Showing phase reversal, in the  
posterior temporal region





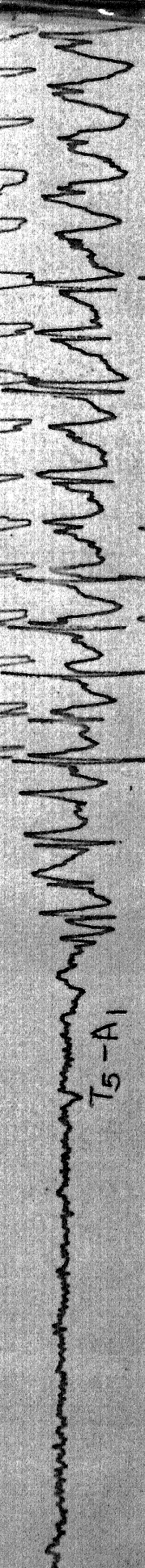
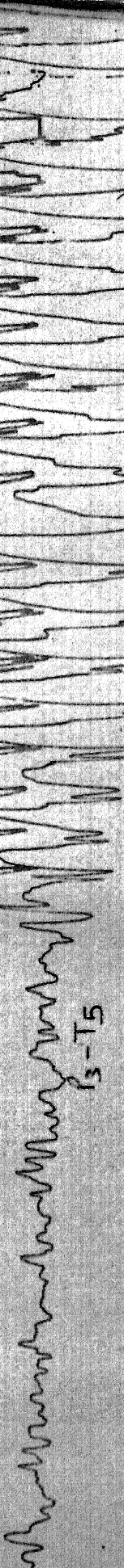
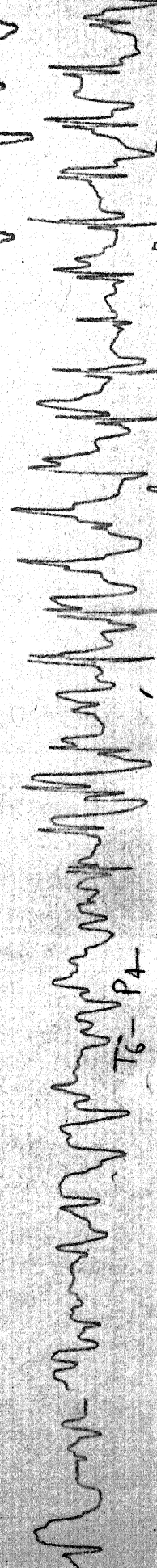
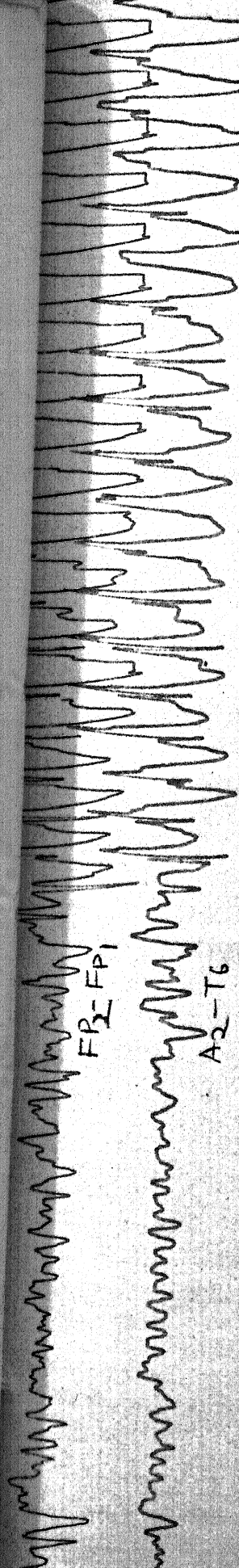


EEG No. 681 : showing generalised polyspikes  
and waves in a case of complex  
partial seizures

50  $\mu$ V

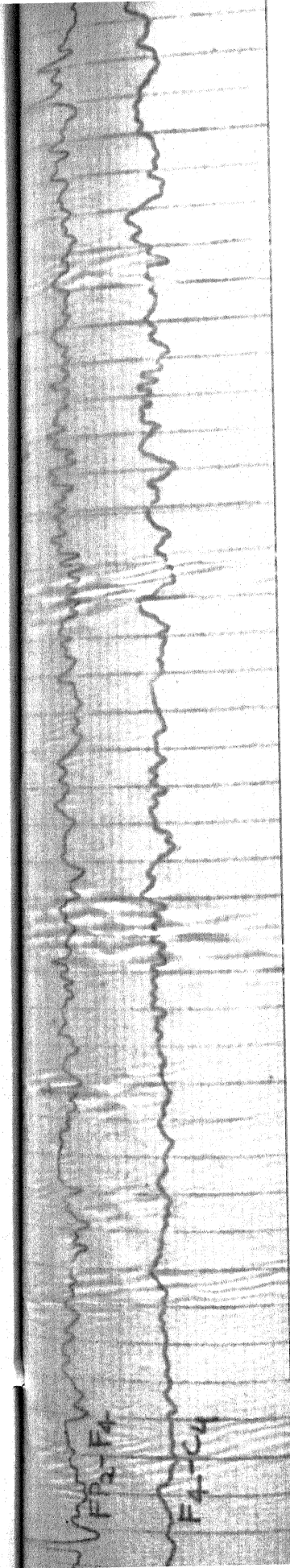


1 Sec.



EEG No. 667 : Showing phase reversal over left  
parietal region.

50  $\mu$ V |  
1 Sec.





F<sub>2</sub>-F<sub>4</sub>

F<sub>4</sub>-C<sub>4</sub>

C<sub>4</sub>-P<sub>4</sub>

P<sub>4</sub>-O<sub>2</sub>

P<sub>1</sub>-F<sub>3</sub>

F<sub>3</sub>-C<sub>3</sub>

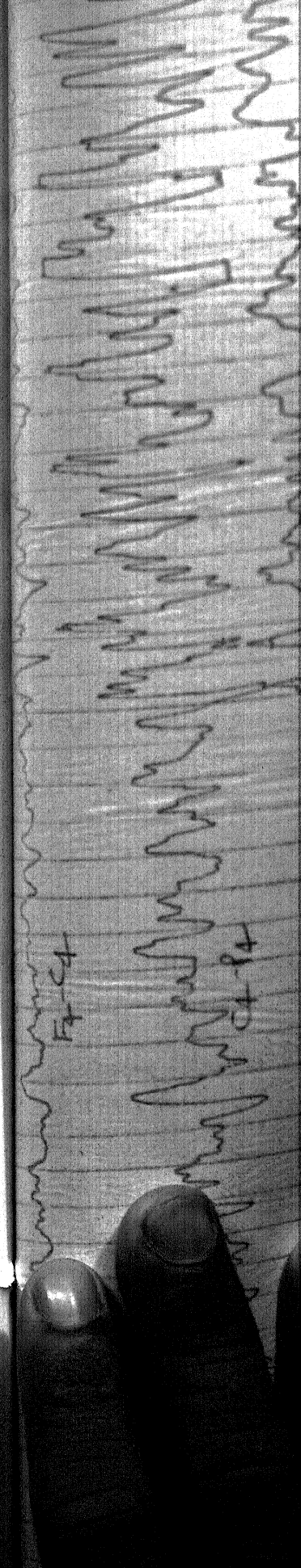
C<sub>3</sub>-P<sub>3</sub>

P<sub>3</sub>-O<sub>1</sub>

G No. 612 : Showing secondary generalisation of  
epileptiform changes starting from  
right side.

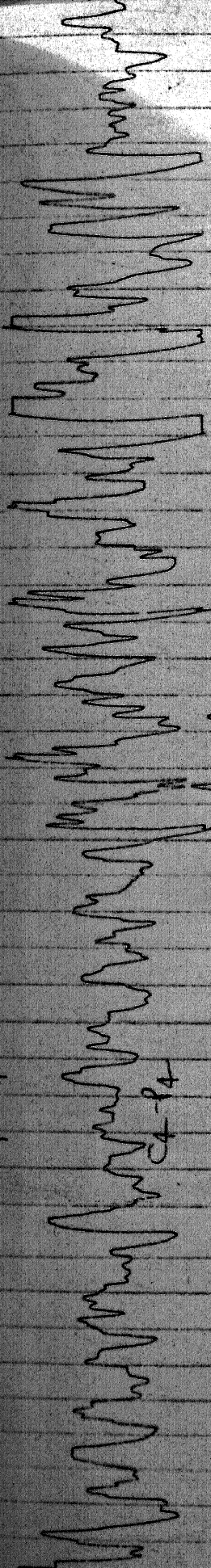
50  $\mu$ V

1 Sec.

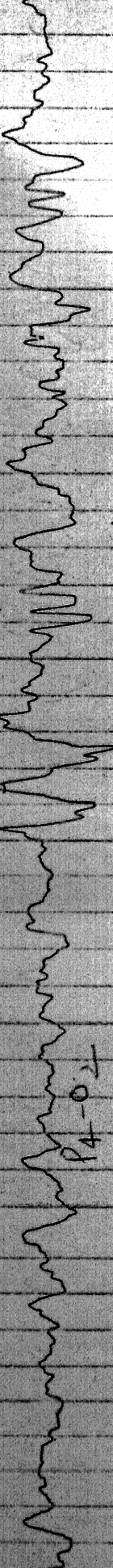




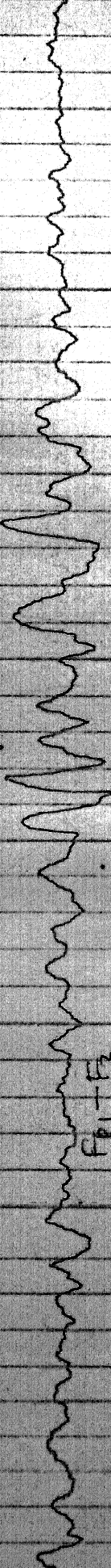
7-4



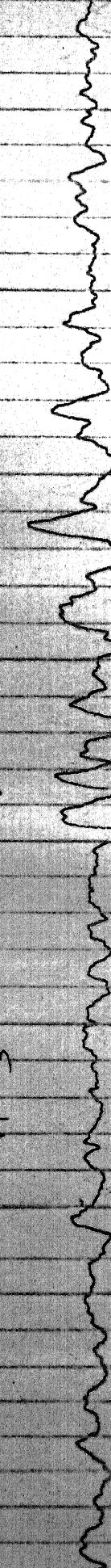
C4-P4



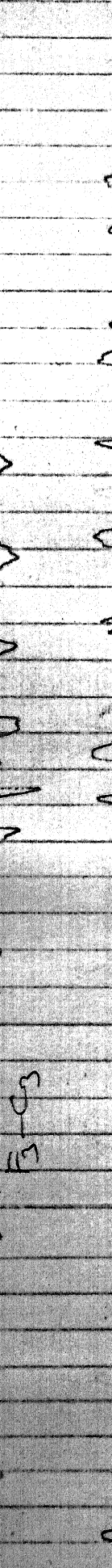
P4-O2



Fp1-F3



F3-C3



C3-P3

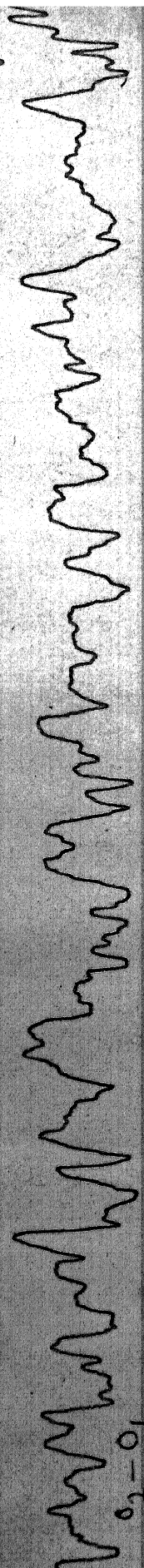
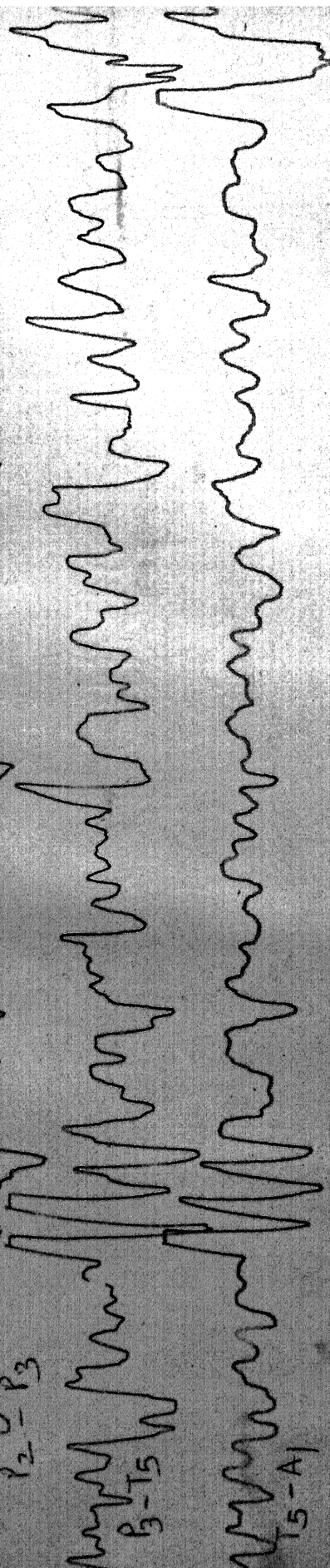
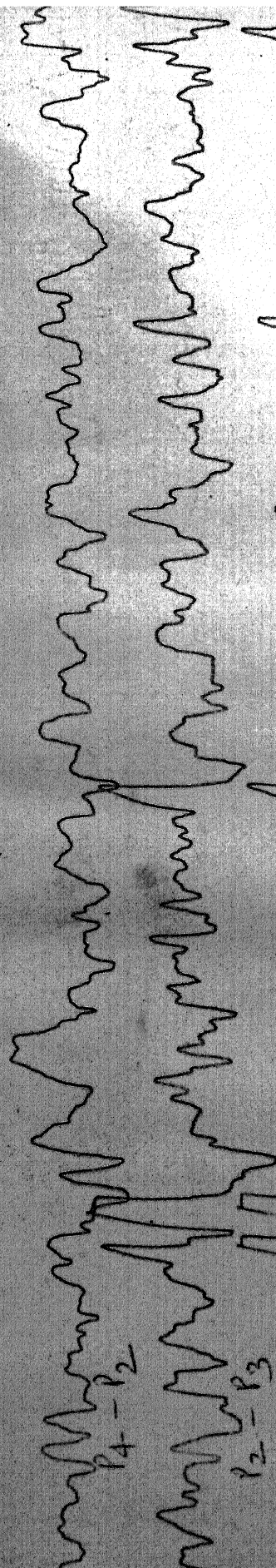
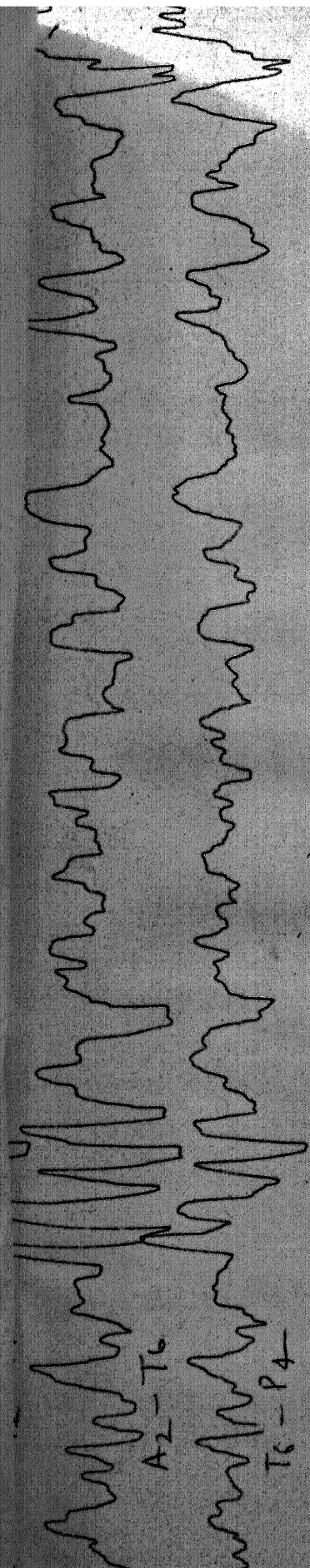


P3-O1

545 : Showing generalised epileptiform discharges  
with maximum amplitude over right parietal  
region in a case of secondarily generalised  
seizures.

50  $\mu$ V | \_\_\_\_\_  
1 Sec.





No. 817 : Showing asymmetry of waves with phase reversal in right frontal region in a case of complex partial seizures.

50  $\mu$ V |  
\_\_\_\_\_

1 Sec.

$F_{P2} - F_8$

$F_8 - T_4$

$T_4 - T_6$

$T_6 - O_2$

$F_{P1} - F_7$

$F_7 - T_3$

$T_3 - T_5$

## **DISCUSSION**

## DISCUSSION

Epilepsy is a common illness. Partial epilepsies are frequent and EEG techniques are very much helpful for diagnosis of partial epilepsies. Considering this we have decided to conduct a correlative study of clinical presentations and EEG abnormalities in cases partial epilepsies.

**Age :** Age specific prevalence ratios for partial epilepsies have been found to be lowest in first decade (Brewis et al - 1966, Juul Jensen 1976 and Heirer et al - 1986). However in this study maximum number of cases belonged to third decade.

Age of onset of seizures was highest in second decade and slightly lower in first decade. Then it decreased substantially in fourth, fifth and sixth decade.

**Sex :** Approximately (70%) of patients were male. Male to female ratio was 2.4:1. Findings were almost similar to Dixit and Mishra (1987-88) and Sood and Mishra (1988-89). Male dominance in this study might be because of more attention given to male member of family in rural population of India.

In many studies males were predominant, male to female ratio being 1.4:1.

**Clinical Seizure types :**

In the present study seizures have been classified on the

basis of classification of epileptic seizures given by ILAE in 1981 and modified in 1982 by Marsden and Reynolds.

Gastaut et al (1975) studied 6000 epileptics and worked out their different classifiable groups and their relative frequency in children and adults. A similar study by Joshi et al (1977) had been carried out in India. Both these studies were based on classification almost similar to that used in the present study. Similar studies were done in this institution by Dixit and Mishra in (1987-88) and Sood and Mishra in (1988-89). Primary generalised seizures dominated our series (55%), in comparison to 30% in the series of Gastaut et al (1975), 20% in the series of Joshi et al (1977), 51.7% by Dixit and Mishra (1987-88) and 62% by Sood and Mishra (1988-89). Next common type was secondarily generalised partial seizures (18%). Figure was higher in comparison to 12% and 15% in Gastaut et al (1975) and Joshi et al (1977) respectively. Cases of simple partial seizures in the present study were (15%) as compared to 50% (Joshi et al, 1977). Smallest number of cases were of complex partial seizures (7.1) which was almost similar i.e. 7% in the study of Joshi et al (1977).

#### FEATURES OF SECONDARILY GENERALISED PARTIAL SEIZURES

In the present study, out of 196 cases, 36(18.36%) cases were of this type of seizures premonitory symptoms were present clinically, in 16 cases.



Six cases presented clinically as tonic spasms, while in 30 cases, clinical presentation was of tonic clonic types. In a study done by Dixit et al (1987-88) these cases were included in primary generalised epilepsy. In these particular cases, it is thought that the spread of focal discharges was so rapid, that focal changes were not present clinically but can be recorded in EEG.

Age of patients ranged from 1 month to 52 years. More than One third of patients were below the age of 10 years. Male to female ratio was 2.8:1. Frequency of seizures varied from several attacks in a day to single attack in a year.

Aetiological factor was present in 9(25%) cases, slightly more in comparison to primary generalised seizures. Head injury was comm<sup>o</sup>nest in 4 cases followed by history of birth anoxia in 3 cases and by intracranial space occupying lesion in 3 cases, inflammatory brain disease in 2 cases. In a series by Joshi et al (1977) aetiological factors were present in 60% of cases. The lesser percentage of etiological factors in the present study might be because CT scan could be performed in fewer number of cases, particularly in the suspected cases of intracranial space occupying lesions. According to Gastaut and Garstaut (1976), CT scans detect 20% more cerebral lesions than the combination of long estab<sup>a</sup>lished techniques like, x-ray skull, EEG and cerebral angiography.

Precipitating factors were present in 4 cases (11%) in the form of sleep-4 cases, fatigue - 1 cases, fever - 1 case.

Out of 48 cases, motor symptoms were present in 16 cases (44%). They were present in the form of twitching of angle of mouth, eye movement, clonic movements of limb and tonic spasm. Sensory symptoms were in the form of tingling and numbness sensation 8 (22%) cases. Sensory motor symptoms were present in 10(28%) cases. Autonomic symptoms i.e. epigastric pain were present in 5% cases.

#### EEG FINDINGS AND CLINICAL CORRELATION

EEG abnormalities were detected in about (80%) cases, percentage being significantly higher than in cases with primary generalised seizures. Findings were focal in 7(19.4%) without any secondary generalisation while in 18(50%) cases, secondary generalisation was present. In 4(11.11%) cases no epileptic foci could be detected and findings were generalised and bilaterally symmetrical and synchronous. In 12(33%) cases epileptic focus were on left side and right sided epileptic focus was present in 9(25%) cases. In three cases focus was central. In one another case it was bilateral in both temporal region. This patient was presented clinically with aura of epigastric pain and tonic-clonic convulsions.

Focus was clearly demonstrated in a single lobe area of scalp in 9(25%) cases. In all the cases focus determination was based



In 16(44%) cases, EEG focus involved two areas of scalp i.e. fronto-parietal-2 cases, parieto-occipital 1 case, centroparietal- 6 cases, and occipitotemporal 4 cases, Parietotemporal. EEG changes were spreaded in such a manner that exact localisation was not possible. This might be, because epileptic focus was deep and EEG changes involved two areas of scalp. One patient of head injury showed haematoma in frontal region and EEG showed fronto- parietal focus. Patient had memory impairment and some behavioural abnormality after head injury. One other interesting case, showed central EEG focus, as phase reversal with generalised epileptic discharge bilaterally symmetrical and synchronous. CT scan showed small granuloma in left mid central region. Patient presented clinically with right sided sensory motor, focal onset followed by generalised tonic-clonic convulsions and unconsciousness.

In one case foci were bilateral in both the temporal region. Patient presented clinically with autonomic symptoms as epigastric pain before tonic clonic convulsion and unconsciousness. This might be because of mirror foci in two lobes (Kiloh et al, 1982).

There were two patients of tonic spasm, one showed phase reversal in fronto-parietal region with generalised epileptic discharges. Other showed slowing in the fronto-parietal region, with bilateral generalised discharges.

In 4 (11%) cases, EEG showed epileptic discharges, bilaterally symmetrical and almost synchronous without any focal changes. The work of Tukul and Jasper (1952) and Penfield and Jasper (1954) showed it was possible for bilaterally synchronous abnormalities to appear in the EEGs of patients with epilepsy apparently caused by unilateral parasagittal orbitofrontal, or anterior temporal lesions. They called this pattern of bilaterally symmetrical and synchronous discharges due to some focal lesion as secondary bilateral synchrony.

#### FEATURES OF SIMPLE PARTIAL SEIZURES :

In our study, 29(15%) cases were of simple partial seizures. Other studies reported 20% simple partial seizures (Sharan, 1987), whereas 50% and 62% reported by Gastaut et al (1975) and Joshi et al (1977), respectively. The low percentage of simple partial seizures in the present study might be because in Bundelkhand region most of the persons are of rural society and uneducated, did not bother about simple twitching and minor movements in hand or feet without loss of consciousness. Age of the patients ranged from 10 months to 52 years. In approximately half of the cases, seizures started below the age of 10 years. Male to female ratio was 2.2:1.

Predisposing factor were present in (11%) cases. However, Joshi et al (1972) found predisposing factor in 30% of cases, whereas Dixit and Mishra (1987-88) could find predisposing

factor in 15.4% of cases. History of head injury was present in 2(8.69%) cases.

Out of 20 cases, seizures started from left side in 16 cases and from right side in 12 cases. Left sided dominance of symptoms could not be explained. Start of seizure from thumb or fingers could possibly be because representation of hand in brain involves much area and is more prone to injuries.

#### EEG FINDINGS AND CLINICAL CORRELATION

EEG abnormalities could be detected in 16(55.17%) cases, EEG focus involving the single area of scalp was found in 5 cases. One case showed central focus. In 5 cases, EEG focus involved two areas of scalp. In two cases multiple foci was present on one side and exact localisation of focus could not be determined. In two cases EEG focus involved occipito parieto temporal region.

In 7 cases, findings were strictly focal without any generalisation to other side. Whereas in other cases, there is generalisation of epileptic discharges to other side. This might be because epileptic discharges in EEG were spreaded bilaterally but clinically patient did not exhibit generalised features.

#### FEATURES OF COMPLEX PARTIAL SEIZURES

There were 14 cases (7.1%) cases of complex partial

seizures. male to female ratio was 2.6:1. In 7(53.85%) cases seizures started below the age of 10 years. In 16 cases (76.9%) seizures started below the age of 20 years. Findings are almost similar to Aird et al (1967) who reported that 42% cases had their first attack in the first decade and over 75% in the first two decade of life. Visamani and Sawhney (1966) and Reddy (1971) drawn the similar conclusion.

Out of 14 cases, predisposing factors could be worked out only in 3(19.37%) cases in the form of head injury - 1 case and birth anoxia - two cases. This is almost similar with earlier studies done in this institution by Dixit and Mishra (1987-88) and Sood and Mishra (1988-89). Lesser percentage of aetiological factor was probably because of the smaller sample and in expected cases CT scans could not be performed.

Aura was presented in 9(64%) of cases. It is almost similar findings as compared to 60% of cases of Shukla et al, 1979.

Out of 14 cases, 3(21%) case also had tonic clonic convulsions. Several authors included these somatomotor manifestations as a clinical features of psychomotor seizures (Bossi et al, 1984).

#### EEG FINDINGS AND CLINICAL CORRELATION

57.11% cases showed EEG abnormality. Epileptic focus was

present in 8 cases viz. temporal - 3 cases, centrotemporal 1 case, temporo- parietal - 1 case, fronto-temporal - 1 case. In one case foci were bilateral in both the temporal regions and one might be mirror image of the other. Patient presented with somatosensory hallucination, smacking of lips and generalised tonic movements and unconsciousness.

## **SUMMARY AND CONCLUSIONS**

## SUMMARY AND CONCLUSIONS

---

The Present study entitled "A correlative study of clinical presentations and electroencephalographic abnormalities in cases of partial epilepsies" was carried out on 196 patients of various types of epilepsies. A detailed history was taken from patients and relatives. Detailed physical examination with more stress on examination of nervous system was done in every case. Various laboratory investigations including routine EEG was done in every case. CT scans were done whenever possible. Observations were tabulated and analysed.

5% of cases were of unclassified epileptic seizures. In nearly half of cases seizures started in the first decade. Overall male to female ratio was 2.4:1. Approximately 55% of cases belonged to the group of primarily generalised seizures, and 40% to the group of partial seizures. Amongst partial seizures, secondarily generalised partial seizures were most common 36 (45%) cases, followed by simple partial seizures 29 (36.7%) cases, and complex partial seizures 14 (17.7%) cases.

58 (74.3%) patients presented with history of recurrent seizures. 13 (16%) cases presented with history of only single seizure episode. 7 (9%) cases presented as status epilepticus. One patient (1.26%) presented as chronic epilepsia partialis continua.

Age of patients ranged from 6 months to 52 years. Seizure frequency was variable. In nearly half of cases seizures started below the age of 10 years. Predisposing factors were present in about 12(15%) cases they consisted of sleep, sleep deprivation, fatigue and fever mainly. Premonitory symptoms were present in 31% of cases, mainly in cases belonging to the group of secondarily generalised partial seizures and complex partial seizures. Ictal events consisted of unconsciousness 31(39.2%) cases, motor phenomena 55(69.6%) cases, Sensory involvement 20(25.3%) cases & autonomic features in 4(5%) cases.

Post ictal features were present in 38(48%) cases. They mainly consisted of Headache, drowsiness, postictal paresis, generalised weakness, confusional states and combination of these.

Associated neurological problems were present in 12(15%) cases. They consisted of mental retardation in 3 cases, hemiparesis in 3 cases, transient inability to speak in 2 cases, Hyperactivity in one case, optic atrophy in one case, confusional state in one case and post ictal paresis in one case each. Respiratory system involvement was present in 2 cases in form of pulmonary tubercular infiltration (1 case) and primary complex in 1 case. EEG abnormalities were present in 53 cases of partial Seizures out of total 79 cases of partial Seizures, and in 56 cases of primarily generalised Seizures out of 108 cases of generalised Seizures.



Thus EEG abnormalities were observed in 67% cases of partial Seizures and in 52% cases of primarily generalised seizures.

CT scan could be performed in a limited number of cases (10 cases). It was abnormal in 3 cases with partial Seizures. In one case it showed a parietal lobe granuloma on left side. In a single case a tuberculoma in central region was present. In Rest of cases CT scans were normal.

Following conclusions could be drawn from the present study.

1. Patients were of younger age group and seizures started below the age of 20 years in most of the cases.

2. Primarily generalised Seizures were commonest type of seizures (55%). Partial Seizures comprised nearly 40% of cases amongst all epilepsies.

3. Males predominated the sample as male to female ratio was 2.4 : 1.

4. The commonest type of partial seizures were secondarily generalised partial seizures (45.6%) followed by simple partial seizures (36.7%) and complex partial seizures (17.7%).

5. Motor phenomena were the commonest (69.6%) components of ictal events followed by sensory involvement (25.3%) and autonomic features (5%).

6. EEG abnormalities were present more commonly in partial seizures (67%) as compared to that in primarily generalised seizures (52%).

7. The common EEG abnormalities in partial seizures were, sharp waves, burst of sharp waves and high voltage sinusoidal waves.

8. EEG morphological characteristics had little correlation with different types of partial seizures(except complex partial seizures) but topographical distribution of EEG abnormalities correlated well with site of seizure onset.

9. Localisation of epileptic foci to a specific region was possible in 17 cases out 79 cases(21.5%) on routine EEG.

## BIBLIOGRAPHY

## BIBLIOGRAPHY

---

1. Aircardi J : Childhood epilepsies with brief myoclonic, atonic or tonic seizures, part 3, 92-93 : A text book of Epilepsy, Edited by John Laidlaw and Alan Richens. Second Edition, Churchill Livingstone, Edinburg, 1982.
2. Aircardi J : The problem of the Lennox syndrome. Developmental medicine and Child Neurology, 15 : 77:1973.
3. Aird RB, Benturini AM and Spielman PM: Arch Neurol, 16: 67: 1967.
4. Ajmone Marsen C : Electroencephalographic aspects of Epileptic. Neuronal Aggregates: Epilepsia, 2: 22 1961.
5. Andermann F, Robb JP : Absence status a appraisal following review of thirty eight patients. Epilepsia. 13 : 177-182, 1972.
6. Annergers JF, Garbow JD, Croover RV, Laws ER, Elveback LR and Kurland LT : Seizures after head trauma : a population study. Neurology (Minneapolis) 30 : 683 , 1980.
7. Blume WT, David RG, Gomez MR : Generalized sharp and slow wave complexes : associated clinical features and long term follow up. Brain, 96 : 289-306, 1973.
8. BO Hjorth : Localization of foci in the scalp field. Petersen Raven press, New York, 483-492, 1976.
9. Bogdonoff BM, Stafferd CR, Green L, Connzair CF, CT evalution in patients with focal epilepsy. Neurology (Minneapolis), 25: 1013-17, 1975.

10. Brown JK, Fits in children : In a text book of epilepsy (Ed. J. Laidlaw and A Richens) 2nd edn. 34. Churchill Livingstone, Edinburg. 1982.
11. Brown JK, Purvis RJ, Forfar JO, Cockburn P: Neurological aspect of perinatal asphyxias. Develop Med, Child Neurol, 16 : 567-580, 1984.
12. Burr W, Steform H, Penin H : Epileptic activity during sleep and wakefulness. Eur Neurol, 25(Suppl) 141-145, 1986.
13. Caffi J, Zur Frege Klinischer : An falls formen bei psychomotorischem epilepsie. Schwees Med Mocheruch, 103 : 469-75, 1973.
14. Chandy J, Mathai KV, Abraham K, Jacob JC and Rachhawat BK : Preliminary report to ICMR on project No. URA-IND Vol. 4, ICMR, New Delhi, p. 66, 1966.
15. Chenric JJ, Aireardi J : Childhood epileptic encephalopathy with slow spike wave : a statistical study of 80 cases. Epilepsia, 13 : 259-271, 1972.
16. Chen RC and Forster FM : Currive epilepsy and gelastic epilepsy. Neurology (Minneapolis), 23:1019+ 1973.
17. Commission on classification and Terminology ILAE proposed revision of clinical & EEG classification of epileptic seizures - Epilepsia 1982; 22:480-501.

18. Commission on classification and terminology ILAE proposal for classification of epilepsies & epileptic syndromes, *Epilepsia* 1985 : 26, 268-78.

19. Commission on classification & terminology ILAE proposal for revised classification of epilepsies & epileptic syndromes *Epilepsia* 1989:30-38998.

20. Coveness MF, Walker AI and Ascroft PB : Incidence of posttraumatic epilepsy in Korean Veterans as compared with those from world war I and II. *Journal of Neuro-surgery*, 19 : 122, 1962.

21. Critchley MR. EEG today. *J. Roy Soc Med*, 71:473-76, 1976.

22. Crombie DL, Gross KM, Fry J, Pinsent RJ and Watts CA, A survey of epilepsies in general practice A report by the Research Committee of college of general practitioners. *Br Med J*. 2:216+422, 1960.

23. Currie S, Neathfield KWG, Henson RD and Scott DF : Clinical course and prognosis of temporal lobe epilepsy. A survey of 666 patients. *Brain*, 94:173, 1971.

24. Dalby MA : Epilepsy and three per second spike and wave rhythms. *Acta Neurologica Scand*, 45 (Suppl), 40:173, 1969.

25. Dena-Heeri J. Trimble MR and Oxley J : prolactin and Gonadotrophin changes following generalized and partial seizures. *J Neurol Neuro Surg Psychiat*, 46, 331, 1983.

26. Deniel E. Jacome : Temporal Lobe syncope : Clinical Variants. *Clinical Electroencephalography*, 20(1):58-65, 1989.

27. Delgado-Escueta AV, Buresal P, Enrile, Triemen DM : Complex partial partial seizures on closed circuit TV and EEG : a study of 691 attacks in 79 patients. Ann Neurology 11 : 292-300, 1982.
28. Do Jong RN : Psychomotor or temporal lobe epilepsy. A review of the development of our present concepts. Neurology. 7 : 1, 1957.
29. Dennerll RP : Cognitive deficits and lateral Brain dysfunction in temporal lobe epilepsy, Epilepsia 5:177, 1964.
30. Dennis BS, Robert DS, Herman F. et al: A reliable method for localising deep intracranial sources of the EEG. Neurology, 35, 1702-1707, 1985.
31. Desai AD: Psychomotor epilepsy. Neurology India. 16 : 18, 1968.
32. Dewhurst K and Dear AN : Sudden religious conversions in temporal lobe epilepsy. Brit J Psychiat. 177:497-507, 1970.
33. Dixit SS : Clinical and electroencephalographic study of epilepsy in Bundelkhand. Thesis submitted for M.D. (Med) Bundelkhand University. 1989 (Unpublished).
34. Dodge PR, Richardson EP Jr and Victor M : Recurrent convulsive seizures as a sequel to cerebral infarction. A clinical and pathological study. Brain. 77:610, 1954.
35. Doose H, Gerken H, Leonardt R, Volzke E and Vols C : Centro-encephalic myoclonic-astatic petit mal. Clinical and genetic investigations. Neuropediatric, 2 : 59, 1970.

36. Duvoisin RC and Vinson WM: Tuberous sclerosis :report of three cases without mental defect.Journal of the american Medical Association,175 : 869,1961.
37. Eccles JC : Postsynaptic inhibition in the central nervous system.In the Neurosciences.A study program p.408,Ed. by G.C.Quatton.T Melnechuk and poschim H,New York.Rock Feller University,Press.
38. Efron R : Postepileptic paralysis.Theoretical functional anatomy of the human brain.Little Brown,Doston Massachusetts,(1961).
39. Efron R : Posteptic paralysis : Theoretical critique and report of a case.Brain,84 : 381,1961.
40. Engel J,Driver MV and Falconer MA : Electrophysio-logical correlates of pathology and surgical result in tomportal lobe epilepsy.Brain,98 : 129,1975.
41. Esquird JED :Quoted by Dowhurst and Sear.1970 (1836).
42. Falconer MA and Taylor DC :Temporal lobe epilepsy : Clinical Features,pathology,diagnosis and treatment,in the modern trends in psychological medicine (2) Harding Price,London, Dutterworths, 1970.
43. Fox RH,wilkins DC : Spontaneous periodic hypothermia. Diencephalic epilepsy.Br Med J.2 : 293,1973.
44. Gastaut H : CT in epilepsy.Epilepsia,17:337-38,1976.



45. Gastaut H and Fischer-williams N : The physiopathology of epileptic seizures.Epilepsia Amsterdam,10 (Suppl). 1-28,1960.
46. Gastaut H,Gastaut JL,Conclaves silva CE and Fernandez sanchez CR : Relative frequency of different types of epilepsy.A study employing the classification of the International league Against Epilepsy.Epilepsia. 16 : 457-467,1975.
47. Gastaut H and Fischer-williams M : The physiopathology of epileptic seizures.In Handbook of physiology Ed.by J Field,HW Magoun and VE Hall (b),P.24,Washington,American physiological Society,1959.
48. Gastaut H : Comment on "Petit mal variant revisited "Epilepsia, 12 :97-99,1971.
49. Gastaut H : Dictionary of epilepsy Part I : Definitions, Geneva, WHO,1971.
50. Gastaut H,Roger J.Sonlayrol R et al : Childhood epileptic encephalopathy with diffuse slow spike waves(Otherwise known as petit mal Variant) or Lennox syndrome.Epilepsia.7:139-179,1966.
51. Seiber S,Bancard J,Talairach T,Bonis A,Engelvin M end Horsard-Bouchand H: Automatisms during frontal lobe epileptic seizure.Brain,99 :447,1976.
52. Gibberd FB: The prognosis of petit mal.Brain,89 : 531- 538, 1966.
53. Gibbs FA : Abnormal electrical activity in the temporal

54. Gibbs FA & Gibbs EL :Atlas of electroencephelography. Vol II,Epilepsy,2nd Edn.Reading : Addison,Westey,1952.
55. Gibbs FA & Gibbs EL : Atlas of electroencephalography.Addison Wesley Press Inc.Cambridge Mess,1964.
56. Gibbs FA & Gibbs EL : Atlas of EEG reading Narsachaseth, Addision- Wesley,Publishing Co.,1952.
57. Gibbs FA and Gibbs EL and Lennox WG :Epilepsy : A paroxysmal cerebral dysrhythmia.Brain,60 : 377,1937.
58. Gibbs FA,Davies H and Lennox WG : The EEG in epilepsy and in conditions of impaired consciousness.Archieves of Meurology and Psychiatry (Chicago). 34 : 1133,1935.
59. Gibbs EL,Gibbs FA and Fuster B : Psychomotor epilepsy.Arch Neurol Psychist.60 : 331,1948.
60. Gloor,Rsai and Haddad :An assessment of the value of sleep electroencephalography for the diagnosis of temporal lobe epilepsy.Electroencephalography and clinical neurophysiology,10 : 663,1958.
61. Gopalkrishanan PN,Lal S.Murli Mohan Rao J : Narayan Rao S.Ramschandran V and Mani KS : Epilepsy (Part I): A retrospective clinical analysis of over 1800 cases. Meurology,India, 16 : 57-61, 1968.
62. Grudainaka B : Epidemiology of epilepsy in populaion of a large industrial city.Incidence and prevalence(in polish)

neurologia neurochirurgia Polska,8,175,1974.

63. Gumpert J.Hensotia P and Upton A :Gelastic epilepsy,J Neurol,Neurosurg.psychiat. 33 : 479,1970.
64. Hearer Dallas W.Anderson and Bruce S Schoenberg : Prevalence and clinical feature of epilepsy in a Biracial United States Population.Epilepsia,27(1) : 66-75,1986.
65. Halasz P : Sleep arousal and EEG manifestations of generalised epilepsy with spike wave pattern. Elsevier.Amsterdam,97-107,1984.
66. Hanser WA and Kurland LT : The epidemiology of epilepsy in Rochester,Minnesota,1935 through 1967,Epilepsia.16 : 1,1975.
67. Hierons R and Seunders M : Impotence in patients with temporal lobe lesions.Lancet,2:761,1966.
68. Hyllested K and pakenberg H : Prognosis in epilepsy of late onset. Neurology (Minneapolis), 13: 641, 1963.
69. Jeavons PM, Clark JE and Maheshwari MC : Treatment of generalised epilepsies of childhood and adolescence with sodium vaiproate (Epilim). Developmental Medicine and child Neurology, 19; 9 : 1977.
70. Jesper HH : Electrical signs of epileptic discharges. Electroencepha. Clin Neurophysiol, 1 : 11-18; 1949.
71. Jesper N and Droogleever-Fortuyn J : Experimental studies on

the functional anatomy of Petit mal epilepsy. Research Publications of the Association for Research in Nervous and Mental Disease, 26 : 272; 1947.

72. Jenett WB : Early traumatic epilepsy. Definition identify. Lancet, 1 : 1023; 1969.

73. Jennett B, Teacher D and Dennie S : Epilepsy after head injury. Residual risk after varying fit free intervals since injury. Lancet, 2 : 652; 1973.

74. Jennett WB : Predicting epilepsy after blunt head injuries. Br. Med J, 4 : 1215; 1965.

75. Jeavons PM : Nosological problems of myoclonic epilepsies in childhood and adolescence. Develop Med Child Neurol, 19 : 3; 1977.

76. Jeroma Engle Jr, David E Kuhi, Phelps ME et al : Comparative localization of epileptic foci in partial epilepsy by PCT and EEG. Annals Neurol, 12 (6) : 529; 1982.

77. Jesus Calleja, Rosario Carpizo and Jose Berciano : Orgasmic epilepsy. Epilepsia, 29(5): 635-639 : 1988.

78. John R. Hughes and Gregory Gruener : The success of EEG in confirming epilepsy revisited. Clinical Electroencephalography, 16(2) : 98-103, 1985.

79. Joseph MW and Richard PB : Periodic lateralised epileptiform discharges - Long Term outcome in adults. Epilepsia, 28(5) : 533-536; 1987.

80. Joshi VD : Clinical and electroencephalographic studies in epilepsy. m.D. (Med.) Thesis, submitted to Banaras Hindu University, 1973.
81. Juul-Jensen P: Epilepsy : A clinical and social analysis of 1020 adult patients with epileptic seizure, Acta Neurol scand, 40 (Suppl, 15) 1 - 148; 1964.
82. Juul-Jensen P : Epilepsy : A clinical and social analysis of 1020 adult patients with epileptic seizure, Acta Neurol scand, 40 (Suppl, 5) : 1-285; 1964.
83. Juul-Jensen P, Ipsen J : Prevalence and incidence of epilepsy in greater Aarhus. Epileptology, proceedings of the seventh international symposium on epilepsy. Berlin/West, June 1975, Ed. Jain D, p. 10, Stuttgart, Georg Thieme Publishers, 1976.
84. Kalisperumal VG, Sundararaj M and Mani KS : Seizure prognosis for partial epilepsies in India, Epilepsy Res., 3: 86-91, 1989.
85. Karagulla S and Robertson RE : Psychical phenomenon in temporal lobe epilepsy and the psychosis. Brit Med J, 1 : 748; 1955.
86. Kellaway P :The incidence significance and natural history of spike foci in children. Current clinical Neurophysiology, Hollend, 151-75;1981.
87. Kiloh LG, MC Comar AJ and Gaselton JN : Clinical : EEG, 3rd Edition. Butlerworths, 85 : 94 : 1972 .

88. Kiloh LG, MC Comar AJ, Osselton JW and Upton ARM : Clinical electroencephalography, 1981.
89. King AW and Ajmone Marson C : Clinical features and ictal patterns in epileptic patients with E.E.G. temporal foci. Annals Keurology, 2 : 1-38:1977.
90. Klass DW : EEG manifestation of complex partial seizures. Advances in Neurology, Vol. 11 : 113-140, 1975.
91. Komsuoglu, SS, Ozmenoglu M, Ozcan C and Gurhan H: Complex partial status epilepticus. Clinical Electroencephalography. 19(3): 167 - 171, 1988.
92. Kriebel J and schleger M : EEG following sleep deprivation : Important diagnostic tool in epileptic seizures. Zeitschroft fur Neurologie. 205 : 193, 1973.
93. Kruse R : Das myoklonisch-sstatische Petit mal. Berlin, Springer, 1968.
94. Loges JC and Gomez MR : Tuberous sclerosis re-appraisal of clinical entity. Mayo Clinic Proceedings. 42 : 26:1967.
95. Laidlow and Richens A : A text book of epilepsy, 2nd edition, Churchill Livingstone, Edinburg, 1982.
96. Lennox WG, Davis JP : Clinical correlates of the fast and slow wave electroencephalogram. Trans Am Neurol Assoc. 74 : 194-97, 1949.

97. Lennox WG : Epilepsy and related disorders. Boston, Little Brown and Co., 1960.
98. Lennox WG : President's address association for research in Nervous and Mental Disease. Proceedings, 26: XV-XIX, 1947.
99. Lennox WG, Gibbs EL and Gibbs FA : Inheritance of cerebral Dysarrhythmia and epilepsy. Archives of Neurology and Psychiatry. 44: 1155, 1940.
100. Lugaresi E, Passaglia P, Frank L et al : Evaluation and prognosis of Primary generalized epilepsies of the petit mal absence type p. 1-20, 1973.
101. Majid Malale : Mid-line epileptiform activity in adult electroencephalograms. Clinical Electroencephalography. 16(1) : 54-61, 1985.
102. Markand CN : Slow spike-wave activity in EEG and associated clinical features often called "Lennox" or Lennox-Gastaut" syndrome. Neurology (Minneapolis). 27: 746-757, 1977.
103. Maradon CD and Reynolds EH : Neurology : In a text book of epilepsy (ed. J Laidlaw and Richens A) 2nd edn. p. 132. Churchill Livingstone, Edinburg, 1982.
104. Manuel R. Comex MD and Donald W Class MD : Epilepsies of infancy and childhood. Ann Neurol. 13 : 113-24, 1983.
105. Mathei KV : Investigation into methods for the

rehabilitation of persons disabled by convulsive disorders. Final report, S.R.S. project No.19-p-58113.F-01 department of Neurological sciences, Christian Medical College and Hospital, Vellore, South India, p.100.1971.

106. Mathicson G : Pathology : In a text book of epilepsy (ed. J Laidlaw and Richens A) 2nd edn.p.437, Churchill Livingstone, Edinburg.1982.

107. Mathews WS : The use & abuse of EEG. Lancet, ii:577-79, 1964.

iii. Matauo y and pcnott JR : Focal positive spikes in EEG. Electroencephal and Clin Neurophysiol, 42 : 15, 1977.

108. Meldrum BS : Pathophysiology : In a text book of epilepsy (edn. J Laidlaw and A Richens) 2nd edn.p.456, Churchill livingatone, Edinburg. 1982.

109. Merlis JV, Grossman C and Henriksen GF : Comparative effectiveness of sleep and Ht-vazol activated EEG. Electroencephal. & Clin Neurophysiol, 3 : 71, 1951.

110. Miriam Y. Neufeld Dan F Cobn and Amos D Korczyn : Sphenoidal EEG recording in complex partial seizures. Clinical electroencophalography, 17(3): 139-141, 1986.

111. Mohan PK : Clinical and electroencephalographic studies in temporal lobe epilepsy. M.D. (Med) Thesis submitted to Banaras Hindu University. 1974.

112. Mourice-William RM : Micturition symptoms in frontal lobe tumours. Jr Neuro. Surg. & Psychi. 37 : 431, 1974.



113. Niedermeyer E : The Lennox-Gastaut syndrome : A severe type of childhood epilepsy. Deutsche Zeitschrift für Nervenheilkunde. 195 : 263, 1969.
114. Niedermeyer E and Rocca U : The diagnostic significance of sleep EEG in temporal lobe epilepsy. European Neurol. 7:119:1972.
115. Offen ML, Devidoff RA, Troof BT and Richey ET : Dacrystic epilepsy. J Neurol. Neurosurg. & Psychi. 39 : 829:1976.
116. Cgden TE, Aird RB and Garoutte BC : The nature of bilateral and synchronous cerebral spiking. Acta Psychi. Neurol. Scand., 31: 273, 1956.
117. Paolo P, Roberto DA, Giovanni A and Elio L : Drop attacks: An ominous change in the evolution of partial epilepsy. Neurology. 35 : 1725-1730. 1985.
118. Parker HL : Epileptiform convulsions. The incidence of attack in cases of intracranial tumour. Archives of Neurology and Psychiatry (Chicago). 23 : 1032:1930.
119. Penfields W, Erickson TC and Tarlov I : Relations of intracranial tumours and symptomatic epilepsy. Archive of Neurology and Psychiatry (Chicago), 44 : 300, 1940.
120. Penfield W and Kristianson K : Epileptic seizure pattern. Thomas Springfield, 111:1951.
121. Penfield and Jasper H : Epilepsy and the Functional anatomy of the human Brain. Little Brown Boston, Massachusetts, 1954.

122. Penry JK and Daly DD : Complex partial seizures and their treatment, advances in Neurology. Vol.11. Raven Press, New York, 1975.
123. Pratt KL. Mattson RH. Weilers NJ and Williams R : EEG activation of epileptics following sleep deprivation. a prospective study of 114 cases. EEG and Clin. Neurophysiology, 24:11, 1968.
124. Prince DA and Wilder BJ : Control Mechanisms in Cortical epileptogenic foci, " Surround Inhibition" Archives of Neurology, 16:194. 1967.
125. Phillips CG : Action of antidromic pyramidal volleys on single Betz cells in the cat. Quarterly Journal of experimental physiology, 44:1, 1959.
126. Ramamurthi B and Gurunathan SK : Epilepsy in Ayurveds. Neurology India. 17: 91-92, 1969.
127. Rasmussen. Olszewski J and Smith D: Focal seizures due to chronic localized encopelitis. Neurology ( Minneapolis). 8 : 435, 1958.
128. Raynor RB, Paine RS and Cermichael EA : Epilepsy of late onset. Neurology ( Minneapolis). 9:111, 1959.
129. Reddy GC : MD(Psychiatry) Thesis, Bangalore University. 1971.
130. Roberto G. Angela M. Sebastiano L. Paolo P. and Elio L : Epilepsy presenting as memory disturbances. Epilepsia. 29(5):624-629:1988.

131. Roger J. Dranet C, Menendez P, Bureau M : Less epilepsies particles de L ienfant, evolution et factures do prognostic Rev EEG Neurophysiol.11 : 431-7, 1981.
132. Scollo-Lavizzari G, Prolli : Activation effects of sleep deprivation and sleep in seizure patients. An EEG study. Eur. Neurol. 13 : 1, 1975.
133. Schineider N, Varsella F, Karbowski K : The Lennox syndrome : a clinical study of 40 children. Eur. Neurol 4 : 289-300, 1970.
134. Sheehan S : One Thousand cases of late onset epilepsy. Irish Journal of Medical Sciences, 6:261, 1958.
135. Shimazezone Y, Krai T, ckuma : Disturbance of consciousness in Petit mal epilepsy. Epilepsia. 2 : 49:1953.
136. Shorvon SD : The epidemiology of epilepsy. Neurology India. 35 : 271-74, 1987.
137. Shukla D and Ammoumi AA : Electro cerebral silence and nasopharyngeal EEG. Clinical Electroencephalography. 18(4) : 233-34, 1987.
138. Shukla GD, Katiyar BC, Nigam P : Journal of Indian Medical Association, 73 : 118-123., 1979.
139. Shukla GD and Mishra DN : Journal of Association of physicians of India, 29 : 1981.
140. Shukla GD, Srivastava CN, Katiyar BC : Brit. J. Paych. 134: 288-92. 1979.

141. Shukla GD, Srivastava CN, Katiyar BC, Joshi V and Mohan PK :  
Brit. J. Psychiatry. 135 : 411-417, 1979.
142. Silverman S and Morisaki A : Re-evaluation of sleep EEG. EEG  
and Clin Neurophysiol, 10 : 425:1958.
143. Slater E and Roth M (Ed.) : Clinical  
psychiatry. Bailliere, Tindal and Cosell, London, 1969.
144. Slater E, Beard AW and Glithere E : The schizophrenia like  
psychosis of epilepsy. Brit J psychiat, 109 : 95-150:1963.
145. Soral LL : Epileptic myokinetique q-rave de La premiere  
enfance avec pointe on de Lent (Petit mal Variet) et son  
Traitment. Rev Neurol (Paris). 110 : 215-223 : 1964.
146. Spencer SS, Spencer DD, Williamson PD and Mattson RH : Sexual  
automatism in complex partial seizures. Neurology (Minneapolis). 33  
: 527, 1983.
147. Stevens JR : Paychiatric implications of psychomotor epilepsy.  
Arch Gen Paychiat, 14 : 461-671, 1966.
148. Sumi SM and Teardall RD : Focal seizures : A review of 150  
cases. Neurology (Minneapolis), 13 : 582, 1963.
149. Sutherland JM and Eadie MJ : The epilepsies : Modern  
diagnosis and treatment, 3rd ed. Churchill Livingstone  
Edinburg. 1980.

150. Symonds C : Excitation and inhibition in epilepsy. Brain, 82 : 133, 1959.
151. Taylor DC and Bruton FJ and Covesellis JAN : Focal dysplasia of the cerebral cortex in epilepsy. J Neurol Neuro Surg Oasychiatr, 34 : 369, 1971.
152. Taylor DC : Sexual behaviour and temporal lobe epilepsy. Arch Neurol, 21 : 510, 1969b.
153. Van AW. Binnie CD. Deboer PTE. Venemdebcas W et al : Electroencephalographic findings in antiepileptic drugs trials : A review and report of 6 studies. Epilepsy Res., 1 : 3-15, 1987.
154. Vrich H : In Blackwood W and Corsellis JAN (Eds.) : Green fields Neuropathology. Ch 10. London, Arnold, 1976.
155. Virmani V and Sawhney BB : Neurol India, 14 : 200, 1966.
156. Walter J : Epilepsy. Brain's diseases of the nervous system. Ninth Edition, 613, 1985.
157. William JN, Abdorasool J, W Steven Netzer and Jese Nickols : The anterior temporal electrode in the EEG of the adult. Clinical Electroencephalography. 19(4) : 199-204, 1988.
158. Williams D : The thalamus and epilepsy. Brain. 88 : 539, 1965.
159. Williems D : Temporal Lobe epilepsy. B.M.J., 1 : 1439, 1966.
160. Williams D : The structure of emotions reflected in epileptic experience. Brain, 79 : 29, 1956.